

Phonological Decoding Ability, Spatial Attention, and
Event-Related Potentials

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A handwritten signature in black ink, appearing to read 'Allison Matthews', written over a horizontal line.

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Date 25/7/08

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ABSTRACT

Evidence for a selective spatial attention deficit among children and adults with developmental dyslexia has been interpreted to reflect impairment in the posterior attentional network including the magnocellular-mediated posterior parietal cortex, and particularly the right hemisphere. According to cognitive interpretations, dyslexia stems from a core phonological deficit and it has been shown that phonological decoding is essential for normal reading development. Thus, the aim of the present research was to investigate the relationship between selective spatial attention and reading ability among adults with good and poor phonological decoding ability as assessed by nonword reading ability. Five experiments were conducted to investigate this relationship.

The results of Experiment 1 indicated that poor phonological decoders are slower to search for feature conjunctions, particularly for searches defined by the features of form and motion. These findings are consistent with previous research in populations with dyslexia and suggest differences between good and poor phonological decoders in terms of the guidance of spatial attention. Experiments 2 to 5 aimed to examine differences in attentional processing in more detail by examining the specific mechanisms involved in both sustained and transient spatial attention tasks and their relationship to early event-related potential components. Research has demonstrated that the early sensory components of the event-related potential waveform (P1 and N1) index early visual processing and are modulated during tasks in which attention is manipulated. However few previous studies have investigated the electrophysiological correlates of spatial attention in good and poor readers.

In Experiment 2 the mechanisms of attentional focussing and inhibition were investigated using a task in which a focussing cue preceded a target that was flanked by compatible or incompatible flankers. Poor decoders showed a greater reaction time cost for incongruent stimuli preceded by large cues, which was suggested to indicate difficulty in focussing attention and suppressing information at unattended locations. This finding was

accompanied by a reduction in the modulation of N1 amplitude by both cue-size and flanker compatibility for poor decoders, and a reduction in the modulation of the frontal N2 component by flanker compatibility. Together, these findings suggested hemispheric differences in the functioning of the posterior attentional network as well as differences in inhibitory processing within the frontal attentional network.

Experiment 3 aimed to examine differences between good and poor phonological decoders in the allocation of attention to global and local levels of hierarchical stimuli. Poor phonological decoders were slower than good phonological decoders when attention was directed to both the global and local processing levels. This was accompanied by a lack of task-related modulation of the posterior N1 and N2 components, and an overall increase in N2 amplitude among poor decoders. Together, these findings suggested differences in the early allocation of spatial attention and compensatory processing at later perceptual stages.

Dyslexia has also been associated with performance differences on covert orienting tasks involving valid and invalid spatial cues. These differences are often greater for stimuli presented in the left visual field which is suggestive of a right hemisphere parietal deficit. The aim of Experiment 4 was to investigate covert orienting in good and poor phonological decoders. Poor phonological decoders showed fewer reaction time benefits of valid spatial cues relative to good decoders, particularly for left visual field trials. This effect was greatest for male phonological decoders, who also showed a lack of N1 modulation in the right hemisphere for left visual field trials and an overall lack of attentional modulation of N1 latency. In comparison, female poor decoders showed a greater involvement of the right hemisphere which may reflect compensatory processing due to a left hemisphere deficit.

The aim of Experiment 5 was to investigate the effect of valid and invalid spatial cues on the performance of orthographic and phonological decision tasks. Consistent with early selection models of attention in word recognition, good decoders showed consistent behavioural effects such that reaction time benefits were observed when words were preceded by valid

spatial cues. However, poor phonological decoders showed fewer reaction time benefits for words preceded by valid spatial cues, particularly for words presented in the left visual field when processing was biased towards phonological processing. These behavioural differences were accompanied by an absence of the attentional modulation of both P1 and N1 latency in poor decoders, and this was explained by differences in early perceptual and attentional processing in the posterior attentional network.

Together, the findings of the present series of experiments provide evidence that the spatial attention difficulties observed in developmental dyslexia are also observed in adults who are poor phonological decoders. The poor phonological decoding group generally showed less attentional modulation of the early posterior N1 and P1 components which is consistent with differences in the functioning of the posterior attentional network. The findings of the present research are broadly consistent with the proposal that the phonological decoding deficits observed in developmental dyslexia are associated with attentional processing differences in the posterior parietal cortex. This research also provided preliminary evidence for sex differences in the lateralisation of ERP components which require further investigation.

CHAPTER 1 – READING DEVELOPMENT, WORD RECOGNITION AND DEVELOPMENTAL DYSLEXIA

The aim of this chapter is to review the major cognitive and linguistic determinants of reading ability and reading disability to provide a basis for considering the associated underlying neuroanatomical basis of reading difficulties in Chapter 2. Learning to read requires the development of a system for mapping the visual characteristics of letter and words (orthography) to the sounds that make up spoken words (phonology) and the retrieval of associated verbal and semantic representations. However, fluent reading is also supported by general cognitive processes such as encoding, storage, and retrieval of information from memory, working memory, visual and linguistic coding processes, and meta-linguistic awareness and knowledge (see Vellutino, Fletcher, Snowling, & Scanlon, 2004).

People with developmental dyslexics experience specific problems learning to read, despite average intelligence, the absence of general learning difficulties, sensory deficits, and other factors such as socioeconomic or educational opportunity (Critchley, 1964; Miles, 1993; Snowling, 2000; Stanley & Hall, 1973). Developmental dyslexia occurs in 5% to 15% of the population (Eden & Zeffiro, 1998), is recognised as a neuroanatomical developmental disorder with a genetic component (see Pennington, 1991) and occurs more frequently among males than females (Liederman, Kantrowitz, & Flannery, 2005). Dyslexia typically manifests as extreme difficulty in acquiring the basic skills necessary for word identification, while comprehension for meaning may or may not be impaired. However, dyslexia is also associated with various cognitive deficits (e.g., working memory and naming speed deficits) and a variety of sensory and perceptual problems. In addition, people with dyslexia frequently report visual problems when trying to read, are often impaired on various non-linguistic tasks including auditory, visual, and motor tasks (Stein & Walsh, 1997) and tend to show signs of mild cerebellar dysfunction (Fawcett, Nicolson, & Maclagen, 2001) and abnormal cerebral lateralisation (Miles, 1993). The sensory and perceptual problems experienced in dyslexia are of particular importance to the

current thesis and will be discussed in detail in Chapter 2 in relation to the neuroanatomical basis of reading difficulties.

Models of Word Recognition

Recent definitions of developmental dyslexia emphasise the role of accurate and fluent word recognition (see Lyon, 1995; Lyon & Shaywitz, 2003). Thus, the present review focuses on reading acquisition in the context of models of single word recognition. According to the Dual-Route or Dual-Route Cascaded (DRC) model of word recognition, words can be processed through two relatively independent routes requiring either lexical/orthographic and sublexical/phonological processing (e.g., Coltheart, Curtis, Atkins, & Haller, 1993; Coltheart, Rastle, Perry, Langdon, & Ziegler, 2001). The lexical route proceeds from visual analysis to activation of a mental lexicon in long term memory which contains phonological and orthographic representations of previously learned words. The phonological code for a word can be derived directly from its overall orthographic characteristics or the semantic information retrieved from orthographic characteristics. This processing route allows the reading of familiar words that have an existing lexical representation and irregular words such as 'yacht' that do not follow normal grapheme phoneme conversion rules. In contrast, the sublexical route proceeds from a visual analysis stage to grapheme-phoneme conversion and assembled phonology prior to retrieval of a semantic code. This route is important for decoding unfamiliar words and nonwords (e.g., 'rint' or 'dilt') that do not have a direct lexical representation. The distinction between lexical and sublexical processing is supported by cases of acquired dyslexia in which either the orthographic (surface dyslexia) or phonological (phonological or deep dyslexia) route to reading is disrupted (see Coltheart, 1980; Coltheart, Masterson, Byng, & Riddoch, 1983).

In contrast to the Dual-Route model of word recognition in which either the lexical or sublexical route is activated to produce an assembled phonological code (Coltheart et al., 2001), connectionist models such as the Parallel Distributed Processing (PDP) model (see Harm &

Seidenberg, 1999; Plaut, McClelland, Seidenberg, & Patterson, 1996; Seidenberg & McClelland, 1989) propose a single route to word identification. According to connectionist accounts, phonological and orthographic processes operate concurrently to activate semantic representations of words. The phonological system operates via an auditory, speech based code, whereas the orthographic system provides information about the visual characteristics of words or parts of words. The many connections between input and output nodes allow existing knowledge to generalise to words that have not been seen or taught previously (Plaut et al., 1996). According to this model, grapheme-phoneme conversion rules are not required to read pseudowords or nonwords. Instead a distinction has been proposed between an orthography-phonology and an orthography-semantic-phonology pathway (see Plaut et al., 1996). Indeed, brain imaging research has shown that, although activation in some areas is associated with specific component reading processes, phonological and orthographic processing largely rely on a common language network in the brain (e.g., Rumsey et al., 1997a). The neurobiological nature of reading processes is discussed further in Chapter 2.

Dyslexia: Subtypes or Continuous Abilities?

Whereas some theorists have attempted to explain the heterogeneity of dyslexia by proposing the existence of distinct subtypes, others argue for a continuous ability approach and emphasise the causal role of phonological processing (see Snowling, 2001). Boder (1971) defined dyslexia in terms of either poor eidetic or phonetic skills, and identified three distinct subtypes: dyseidesia, dysphonesia, and dysphoneidesia (mixed). Dyseidesia is a visual/perceptual deficit resulting in problems in perceiving whole words and matching these to auditory representations. People with dyseidesia typically have difficulty reading irregular words and make phonetic regularisation errors in reading and spelling. In contrast, dysphonesia is associated with difficulty in using grapheme-phoneme relationships, resulting in a phonemic/linguistic deficit or speech discrimination deficit. People with dysphonesia typically

have difficulty reading unfamiliar words and may make semantic substitutions in reading (e.g., barn for house). Dysphonia is defined as a combination of deficits in both eidetic and phonetic skills.

Based on the theory that reading development is associated with a shift from visuospatial text analysis mediated by the right hemisphere (RH) to semantic syntactic analysis mediated by the left hemisphere (LH), another typology was developed to classify dyslexia in terms of reading speed and accuracy (Bakker, 1990; Bakker, Moerland, & Goekoop-Goefkens, 1981; Bakker & Vinke, 1985; Masutto, Bravar, & Fabbro, 1994; Vellutino et al., 2004). Children with P-type (perceptual) dyslexia read relatively slowly but accurately, tend to make time consuming errors and show a left ear advantage in dichotic listening tasks. This pattern of difficulties is thought to result from persistent RH reading. In contrast, those with L-type (linguistic) dyslexia read relatively fast but inaccurately, tend to make errors of omission or addition, and show a right ear advantage in dichotic listening tasks. This pattern is thought to result from an untimely shift in reading to the LH. Those with M-type (mixed) dyslexia are both slow and inaccurate readers and make both types of errors.

Another classification based on the dual-route model of word recognition distinguishes dyslexic subtypes on the basis of irregular and nonword reading ability (see Castles & Coltheart, 1993). A deficit in nonword reading, despite normal irregular word reading is thought to represent a sublexical deficit resulting in 'developmental phonological dyslexia'. In contrast, impaired irregular word reading and intact nonword reading is argued to represent impairment in lexical processes resulting in 'developmental surface dyslexia'. These children rely heavily on phonological processing, make regularisation errors and are poor at distinguishing between homophones such as pain/pane or meet/meat. Those who have difficulty with both irregular and nonword reading are defined as having 'mixed' dyslexia. Mixed dyslexia is most common and is also associated with the most severe reading deficits. Several studies have investigated the utility of this typology in discriminating children with dyslexia from control children (e.g., Manis,

Seidenberg, Doi, McBride-Chang, & Petersen, 1996; Manis et al., 1999; Stanovich, Siegel, & Gottardo, 1997). Other research has also shown that adults with a diagnosis of surface and phonological dyslexia show equivalent phonological ability suggesting caution in using this typology for classifying adults with dyslexia (Zabell & Everatt, 2002).

Some researchers argue against the existence of distinct subtypes in dyslexia suggesting that it is more important to explain the variation of reading sub-skills among populations with dyslexia and consider differences between normal readers to be quantitative rather than qualitative in nature (see Griffiths & Snowling, 2002; Olson, Kliegl, Davidson, & Foltz, 1985; Snowling, 1981, 2000; Snowling, 2001; Stanovich & Siegel, 1994; Vellutino et al., 2004). The common definition of dyslexia excludes distal causes such as IQ, educational opportunity, and neurological involvement. The fact that the same distal causes of dyslexia (e.g., poor grapheme phoneme mapping) can occur due to different underlying proximal causes (e.g., poor phonological awareness or type of reading instruction) has been an argument for the broadening of diagnostic criteria so that it is not exclusionary in nature (see Coltheart & Jackson, 1998). Further, diagnosis of dyslexia on the basis of a discrepancy between reading performance and IQ has been criticised on the basis that poor readers across a range of IQs show the same patterns of reading disability, suggesting that reading ability exists on a continuum that includes dyslexia and normal readers (e.g., Stanovich, 1988; Stanovich & Siegel, 1994). Recent definitions of dyslexia have put less emphasis on exclusionary diagnostic criteria and the discrepancy between reading performance and IQ, and emphasise the causal role of a core phonological processing deficit (for a discussion see Lyon, 1995; Lyon & Shaywitz, 2003).

The Phonological Deficit Hypothesis

According to the phonological deficit hypothesis, a weak phonological mechanism and poorly specified phonological representations explain the impairment that those with a diagnosis of dyslexia show on a variety of phonological tasks as well as deficits observed in rapid naming,

verbal learning, and verbal memory (see Bradley & Bryant, 1983; Rack, Snowling, & Olson, 1992; Stanovich, 1988; Stanovich & Siegel, 1994; Wagner & Torgesen, 1987). The ability to acquire alphabetic coding skills and letter knowledge relies heavily on the acquisition of phonological awareness or the implicit and/or explicit awareness that words are composed of individual speech sounds (phonemes) and their combinations (syllables, onsets, rimes) (for reviews see Castles & Coltheart, 2004; Vellutino et al., 2004). Dyslexia is consistently associated with poor phonological awareness as assessed by rhyme detection, and phoneme counting, deletion, and substitution tasks (Snowling, 1981; Stanovich, 1988). In addition, longitudinal studies have reported a causal link between phonological awareness and subsequent success at learning to read (Bradley & Bryant, 1983; Hulme et al., 2002) and training in phonemic awareness has been shown to improve subsequent reading and spelling performance (Bradley & Bryant, 1983, 1985) (for a review see Castles & Coltheart, 2004).

The development of phonemic awareness is thought to facilitate the acquisition of more analytic phonological skills such as phonological decoding or the ability to use grapheme phoneme conversion rules to map from orthography to phonology (Perfetti, 1994). A large number of studies have shown that dyslexia is associated with poor phonological coding ability as assessed by nonword reading tests (Rack et al., 1992; Siegel, 1994) and these difficulties have been shown to continue into adulthood (Bruck, 1993). Phonological and orthographic coding is often assessed using lexical decision tasks in which participants make a phonological decision about which of two nonwords is a homophone of a real word (e.g., kake, dake), or an orthographic decision about which of two homophones is a real word (e.g., rume, room) (Olson et al., 1985; Stanovich & Siegel, 1994; Stanovich & West, 1989). Olson et al. (1985) and Stanovich and Siegel (1994) found that children with dyslexia differed from control children in their ability to perform phonological lexical decision to a greater extent than orthographic lexical decisions.

Despite wide support for the phonological deficit hypothesis, there is also evidence for the interdependency of phonological and orthographic skills. A purely phonological explanation fails to account for cases of dyslexia in which a phonological deficit is not observed or in which orthographic relative to phonological ability is impaired (Castles & Coltheart, 1993; Valdois et al., 2003). According to the ‘severity hypothesis’, the behavioural profile of dyslexia depends on the severity of a phonological deficit in combination with other cognitive abilities, reading experience, and compensatory strategies (Snowling, Gallagher, & Frith, 2003; Valdois et al., 2003). For example, dyslexic readers are often no worse at irregular word reading than reading age matched controls and whereas phonological processing and short term memory contribute unique variance to nonword reading, irregular word reading is predicted by print exposure or reading experience (Griffiths & Snowling, 2002). These findings are consistent with self teaching hypothesis of reading acquisition (Share, 1995, 1999). According to this theory, phonological and orthographic skills are employed on an item by item basis. For example, whereas high frequency or familiar words can be recognised visually with little phonological processing, unfamiliar words can be phonologically decoded using grapheme-phoneme conversion rules. This phonological recoding process is thought to act as a self-teaching mechanism that increases the likelihood of the subsequent development of word specific orthographic representations.

Recent research has shown that rapid automatised naming speed contributes unique variance to reading ability that is not explained by phonological skills, suggesting two independent sources of reading disability (for reviews see Wolf, Bowers, & Biddle, 2000; Wolf et al., 2002). Many dyslexic children show both phonological and rapid automatised naming deficits, and those with a ‘double deficit’ are typically more impaired on reading measures than those with a single deficit (Wolf et al., 2000; Wolf et al., 2002). Naming speed has been found to be associated with orthographic knowledge and reading speed, whereas phonological awareness is correlated with word identification and phonological decoding (e.g., Wolf et al., 2000; Wolf et

al., 2002). However, the causal nature of the relationship between naming deficits and reading difficulties has been questioned (for a review see Vellutino et al., 2004).

Phonological Decoding in Adult Dyslexia

Consistent with the severity hypothesis, some adults are able to compensate for poor reading ability experienced as a child and become relatively skilled readers. For example, dyslexic adults often compensate for poor word recognition skills by drawing on resources such as semantic knowledge, verbal ability, and the use of context (Bruck, 1990; Nation & Snowling, 1998; Snowling, Bishop, & Stothard, 2000; Torgesen et al., 2001). Dyslexics also compensate through the use of analogy and a greater reliance on a visual reading route (Siegel, 1994) and it has been shown that children with better visual memory and slower processing speed are better at non-word reading which may represent two different sources of compensation (Snowling, 2001). Despite development of compensatory strategies, adults dyslexics typically show persistence of some symptoms of reading disability into adulthood (Bruck, 1990, 1992, 1993; Fawcett & Nicolson, 1995; Shaywitz et al., 1999; Zibell & Everatt, 2002). For example, adult dyslexics typically still show impairment on tasks that assess nonword reading and phonological awareness (Ben-Dror, Pollatsek, & Scarpati, 1991; Bruck, 1990; Brunswick, McCrory, Price, Frith, & Frith, 1999; Elbro, Nielsen, & Peterson, 1994; Fawcett & Nicolson, 1995; Gallagher, Laxon, Armstrong, & Frith, 1996; Rack et al., 1992; Shaywitz et al., 1999). In addition, most adult with dyslexia also experience persistent difficulty with spelling (Bruck, 1993; Gallagher et al., 1996; Shaywitz et al., 1999) and require more time to process both words (Miller-Shaul & Breznitz, 2004) and nonwords (Bruck, 1990; Brunn & Farah, 1991; Gallagher et al., 1996; Rack et al., 1992).

Elbro et al. (1994) found that adults with poor phonological coding skills have basic deficits in representing the phonological aspects of words that could not be attributed to factors such as semantic knowledge, phonemic awareness, educational level, and current reading

patterns. Furthermore, differences in comprehension could be explained by differences in phonological coding rather than semantic knowledge. Gallagher (1996) found that some high functioning adult dyslexics performed at the same level as controls on word recognition tests, but performed at a lower level on measures of nonword reading and spelling accuracy, and were slower on spoonerisms, digit naming, and speech rate. These findings suggest the persistence of phonological difficulties in high functioning adults that could not be attributable to cognitive and attentional resources as attested by their academic achievement. Bruck (1993) found that college students with a childhood diagnosis of dyslexia, showed spelling problems that were primarily related to their lack of knowledge of grapheme phoneme mappings.

Summary

Normal reading acquisition requires the mastery of both phonological and orthographic processing skills and current models of word recognition emphasise both phonological and orthographic processing. Recent definitions of developmental dyslexia emphasise the importance a core phonological deficit in the aetiology of the disorder. This phonological deficit is often marked by poor ability to read nonwords or pseudowords and therefore difficulty in reading through the process of grapheme-phoneme conversion or phonological decoding, and these impairments typically continue into adulthood (Ben-Dror et al., 1991; Bruck, 1990). According to sub-type explanations of dyslexia, children with poor phonological processing skills are likely to be diagnosed with developmental phonological dyslexia or dysphonesia, and depending on impairment on measures of orthographic processing, may also be diagnosed with mixed dyslexia or dysphoneidesia. Whether or not dyslexia is classified according to a typology or according to the presence of a core phonological deficit, it is clear that phonological decoding ability is a major determinant of normal reading acquisition and reading disability. The following chapter outlines the major neurobiological explanations for developmental dyslexia, with particular emphasis on the correlates of poor phonological processing skills.

CHAPTER 2 – NEUROBIOLOGICAL EXPLANATIONS OF DYSLEXIA

Several hypotheses have been developed to account for the underlying neuroanatomical basis of dyslexia. A major debate in the neurobiological literature pertains to whether dyslexia represents a perceptual or linguistic disorder or indeed both (for a review see Galaburda, 1999). The phonological deficit hypothesis predicts a linguistic/cognitive basis to dyslexia and implicates areas of the brain that are involved in higher order language processing. These areas include the temporal cortex, inferior parietal lobule and areas of the frontal lobe. Research showing abnormal lateralisation of language function (see Beaton, 1997) and decreased activation in left hemisphere language areas provide support for this hypothesis (see Galaburda, 1999).

In contrast, a sensory and/or perceptual deficit predicts that impairments in areas of sensory pathways cause the linguistic and cognitive deficits observed in dyslexia. These areas include sense organs and thalamic and brain stem nuclei, as well as primary sensory cortices and association areas. Perceptual interpretations include the cerebellar hypothesis (see Nicolson & Fawcett, 1999), the auditory deficit hypothesis (Tallal, 1980), and the visual magnocellular hypothesis (see Lovegrove, 1996). Based on findings of difficulties in processing rapid temporal stimuli in several modalities, it has also been proposed that dyslexia stems from a generalised deficit in processing in several modalities (see Farmer & Klein, 1995).

This chapter briefly reviews the evidence for these major theoretical explanations and focuses at length on visual processing. Numerous lines of converging evidence indicate that dyslexia is associated with a specific abnormality of the magnocellular pathway of the visual system (for reviews see Lovegrove, 1996; Stein, 2001a; Stein & Talcott, 1999). A major challenge for this theory has been to explain *how* a magnocellular deficit causes the pattern of reading deficits observed in dyslexia. One possibility is that abnormalities in early visual processing magnify to produce deficits in processes that are mediated by the Posterior Parietal Cortex (PPC) (see Stein & Walsh, 1997). This theory is supported by research showing deficits

in motion sensitivity, binocular control, eye movements, and spatial attention among dyslexic populations. One theory that has gained empirical support in recent years is that deficient spatial attention mechanisms play a role in the reading difficulties experienced in dyslexia. The relationship between spatial attention and reading ability is discussed at length in Chapter 3.

Atypical Cerebral Lateralisation

The lateralisation of language function to the LH is one of the most robust findings in neuropsychology (for a review see Springer & Deutsch, 1993). Areas of the LH are specialised for language production (Broca's area) and language comprehension (Wernicke's area). In contrast, the RH is specialised for processing nonverbal stimuli such as faces (e.g., Pirozzolo & Rayner, 1977) and its role in language processing is less clear, though some research indicates that the RH is involved in visuospatial processing during ideographic reading that predominates during early reading development (Waldie, 2002). A maturational lag in hemispheric dominance has long been cited as a possible cause of dyslexia (Orton, 1925) and was thought to result in unstable spatial organisation of letters and words. This proposal is consistent with some typical characteristics of dyslexia including mixed hand preference, difficulty with sequencing the days of the week/months of the year, and left/right confusions (Miles, 1993).

Based on findings of an association between left handedness, learning disorders and immune dysfunction, it has been argued that in-utero testosterone levels play a role in the development of dyslexia (Geschwind & Galaburda, 1985). According to the Geschwind-Behan-Galaburda hypothesis, testosterone is involved in the development of language lateralisation, right hemisphere spatial organisation, and right handedness. According to Annett's right shift theory of dyslexia, phonological dyslexia (dysphonesia) results from weak cerebral lateralisation and a shift away from dextrality (Annett, Eglington, & Smythe, 1996). In contrast, surface dyslexia (dyseidesia) is associated with weak representations of words in visual memory, an over-reliance on the LH and therefore a shift towards dextrality. This conception is similar to the

typology proposed by Bakker to account for L-type and P-type dyslexia respectively (see Bakker, 1990). Unfortunately, the relationship between dyslexia and factors such as handedness and immune dysfunction has not always held true when tested empirically or only accounts for a small proportion of the variance in dyslexia (e.g., Hugdahl, Synnevag, & Satz, 1990).

Other evidence of abnormal cerebral lateralisation of language function in dyslexia comes from findings of reversed or reduced asymmetry of the planum temporale and other areas of the brain, behavioural research inferring laterality differences from hemi-field presentation, and remediation studies that have aimed to stimulate the left and right hemispheres of the brain. Abnormalities of the corpus callosum and problems in the interhemispheric transfer of information are also discussed in this chapter. However, perhaps the most compelling evidence for anomalous lateralisation in dyslexia comes from recent neuroimaging research that has consistently shown reduced activation in LH language areas in dyslexia suggestive of a LH linguistic deficit.

The planum temporale is an auditory association area of the temporal lobe, in the Sylvian fissure, posterior to the primary auditory cortex (Heschl's gyrus) and superior to Wernicke's area. Whereas the planum temporale is larger in the LH in about 75% of the population from birth (Shapleske, Rossell, Woodruff, & David, 1999), both post mortem (e.g., Galaburda & Kemper, 1979; Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1985) and brain imaging studies (for reviews see Beaton, 1997; Eckert & Leonard, 2000) (but see Leonard et al., 1993) have shown reduced or reversed asymmetry of the planum temporale in dyslexia. However, whereas symmetry of the planum temporale has been shown to be associated with phonological deficits (nonword reading) (Larsen, Høien, Lundberg, & Odegaard, 1990), phonological deficits have also been observed in groups of dyslexics with normal planum temporale asymmetry (Leonard et al., 1993). Similarly, abnormal planum temporale asymmetry has been associated with abnormalities in the magnocellular layers of the visual system (Livingstone, Rosen, Drislane, & Galaburda, 1991). However, normal planum temporale

asymmetry has also been observed in a group of dyslexics with both magnocellular processing and phonological deficits (Best & Demb, 1999). It is unclear whether the planum temporale symmetry observed in dyslexia reflects a decrease in the size of the left planum temporale or an increase in the size of the right planum temporale (Beaton, 1997), and it has been argued that planum temporale symmetry is more strongly associated with general language impairment rather than specific reading ability (Eckert & Leonard, 2000). For example, in a recent Magnetic Resonance Imaging (MRI) study, dyslexics showed a reduction in the size of the left planum temporale and a significant correlation between dichotic listening performance and planum temporale asymmetry (Hugdahl et al., 2003).

Other research findings suggest differences in hemispheric asymmetries in other areas of the brain. For example, an early Computerised Tomography study, found that a sub-group of dyslexics (with lower verbal IQ scores) showed a reversal of the normal asymmetry observed in parietooccipital areas, such that the RH region was wider than the LH region (Hier, LeMay, Rosenberger, & Perlo, 1978). In another Computerised Tomography study, Haslam et al. (1981, cited in Duara et al., 1991) found that boys with dyslexia showed less left-greater-than-right asymmetry in occipital areas. Further, using MRI, Duara et al. (1991) found that a posterior area of the brain including the angular gyrus was larger in the RH than LH for children with dyslexia and symmetrical in normally reading children.

Abnormal cerebral lateralisation has also been inferred from behavioural research in which stimuli are presented laterally to either the left visual field (LVF) or right visual field (RVF) and are therefore processed first in the contralateral (opposite) RH and LH respectively. Dyslexics often fail to show the same right visual field advantage (e.g., Bloch & Zaidel, 1996; Kershner, 1977; Marcel, Katz, & Smith, 1974; Pirozzolo & Rayner, 1979) and right ear advantage (e.g., Boliek, Obrzut, & Shaw, 1988) that has been observed among normally reading populations and thought to reflect the specialisation of the LH for language. Furthermore, some investigators have reported a LVF advantage for linguistic stimuli in dyslexia, suggesting a

reliance on the visual orthographic RH strategy for reading (Marcel et al., 1974; Waldie, 2002). These findings are consistent with the proposal that acquired deep (phonological) dyslexia is associated with an over-reliance on the RH for reading (Coltheart, 2000, 1980). However, some studies have failed to show reduced laterality or even increased RVF or right ear effects (for a review see Kershner, 1985). For example, Yeni-Komshian, Isenberg, and Goldberg (1975) found that dyslexics had a greater RVF vocal reaction time advantage for verbal stimuli than normal readers, that was largely due to slower responses to LVF stimuli, suggestive of a RH deficit, or disrupted transmission from the RH to the LH. Witelson (1977) found evidence to suggest that dyslexics had typical LH lateralisation but bilateral representation of spatial function which may interfere with the linguistic processing of the LH. It was further suggested that this may lead to over-use of a spatial holistic rather than a linguistic sequential cognitive processing mode. Further, Kershner & Graham (1995) found that the order of presentation affected the right ear advantage in phonological dyslexia, suggesting that an attentional impairment was involved.

According to Bakker (1990), reading development is associated with a shift from RH to LH reading strategies. As previously mentioned, P-type and L-type dyslexia are thought to result from persistent RH reading and an untimely shift to LH reading respectively. The validity of this classification of dyslexia has been supported by various rehabilitation investigations. Bakker posited that P-type dyslexics would benefit from selective stimulation of the LH, whereas L-type dyslexics would benefit from selective stimulation of the RH. Consistent with this hypothesis, visual hemispheric specific stimulation through tachistoscopic presentation of words to each visual hemi-field has been shown to improve reading among dyslexics (e.g., Bakker et al., 1981; Bakker & Vinke, 1985; Lorusso, Facoetti, Paganoni, Pezzani, & Molteni, 2006). However, the mechanisms affected by visual hemisphere specific stimulation have not been well explained and there has been some suggestion that improvement occurs due to non-specific factors such as spatial attention rather than hemisphere specific effects (see Lorusso et al., 2006).

Corpus Callosum Abnormality and Interhemispheric Transfer

Some MRI research indicates that the corpus callosum which connects the two cerebral hemispheres is abnormal in size in dyslexia (for a review see Beaton, 1997) (but see Larsen et al., 1990). Duara et al. (1991) found that dyslexics and particularly dyslexic females had an enlarged splenium of the posterior corpus callosum. Rumsey et al. (1996) also found evidence for an enlarged posterior portion of the corpus callosum (isthmus and splenium) among adult dyslexic men relative to controls. Similarly, Robichon and Habib (1998) found evidence for a larger isthmus among male dyslexic adults and this was found to be related to phonological ability (phonological awareness and nonword reading). The isthmus is thought to connect the temporal and parietal areas of the left and right hemispheres, and a larger isthmus has also been observed among non-right handed males. Robichon and Habib observed that an enlarged isthmus was more common among right handed rather than non-right handed dyslexics, suggesting a difference in the callosal mechanisms involved in dyslexia with LH language representation and bilateral language representation respectively. However, it has also been found that adolescent dyslexics show a reduction in size of the genu (anterior portion) of the corpus callosum, with no differences in the splenium (Hynd et al., 1995). Corpus callosum abnormalities among dyslexics are also consistent with findings of poor phonological processing and nonword reading among those born without a corpus callosum (see Beaton, 1997).

Behavioural research has also implicated inter-hemispheric transfer of information in the aetiology of dyslexia. For example, Gross et al. (1978) found a greater difference between letter duration thresholds in the LVF than RVF among dyslexics relative to controls, suggesting ineffective transfer of information. Dyslexic children have difficulty performing tactile sequencing tasks such as finger tapping and typically perform worse on bimanual relative to unimanual motor coordination tasks (Badian & Wolff, 1977; Gladstone, Best, & Davidson, 1989; Moore, Brown, Markee, Theberge, & Zvi, 1995; Wolff, Cohen, & Drake, 1984; Wolff, Michel, Drake, & Ovrut, 1990) (but see Ramus et al., 2003). For example, Badian and Wolff (1977)

found that dyslexic boys had greater difficulty tapping an alternating hand in comparison to a single hand sequence, particularly in relation to performance of the left hand, suggestive of impaired interhemispheric integration. Support for the role of the corpus callosum in bimanual coordination comes from research showing that patients without a corpus callosum show an inability to perform novel coordinated bimanual movements (Wolff et al., 1990). However, there is also evidence to suggest that LH damage is associated with impairment on bimanual tasks (Gladstone et al., 1989).

Using 'Etch-a-Sketch' bimanual coordination tasks, it has been found that adults with dyslexia show deficits in bimanual coordination particularly when the left hand has to move faster than the right or when the hands had to make mirror image (opposite movements) (Gladstone et al., 1989; Moore et al., 1995). These findings were thought to indicate a problem in the interhemispheric modulation of hemispheric control possibly implicating the contribution of the RH in the control of visuospatial skill (Moore et al., 1995). However, the LH plays an important role in the control of bimanual coordination, suggesting that the problem could lie in LH control of the RH, or in the greater effects of cross-callosal interference on the RH by LH processing (Moore et al., 1995). Gladstone et al. (1989) proposed a model in which the combination of LH dominance for bimanual control, poor interhemispheric communication and anomalous ipsilateral representation could account for the deficits observed in left hand performance in dyslexia.

Other studies have found differences between dyslexics and controls using finger recognition or localisation tasks (e.g., Fabbro et al., 2001; Fletcher, Taylor, Morris, & Satz, 1982; Gross-Glenn & Rothenberg, 1984; Moore, Brown, Markee, Theberge, & Zvi, 1996). In studies in which tactile sequences delivered to one hand are copied using the same (uncrossed) or other (crossed) hand, dyslexics typically perform worse in the crossed hand condition which requires the interhemispheric transfer of information (Fabbro et al., 2001; Summerfield & Michie, 1993) (but see Sotozaki & Parlow, 2006). Patients without a corpus callosum also

perform poorly on crossed trials in interhemispheric transfer tasks and normal children show a developmental improvement on this task with age (Quinn & Geffen, 1986) implicating abnormal development of the corpus callosum in dyslexia. Although Summerfield and Michie (1993) found a relationship between reading ability and performance on the finger localisation task, it was argued that the symptoms of poor interhemispheric transfer are not causal factors in dyslexia but reflect pervasive central nervous system dysfunction. However, Fabbro et al. (2001) found a deficit in callosal transfer in children with L-type and M-type but not P-type dyslexia using a crossed/uncrossed tactile task, and other research has shown that performance on crossed trials in the finger localisation task is related to phonological ability (Moore et al., 1996).

Dyslexics show a greater bilateral advantage (redundancy gain) for visual stimuli when responding with their left hand relative to normal readers suggesting slower left-to-right transfer of information through the corpus callosum (Badzakova-Trajkov, Hamm, & Waldie, 2005). This is partially consistent with a deficit in inter-hemispheric transfer of information as split-brain patients and acallosals also show a greater redundancy gain regardless of response hand. Other studies have measured inter-hemispheric communication more directly by measuring inter-hemispheric transfer time. Using a tactile hemi-field task, Davidson et al (1990) found that language disordered children with a concurrent reading disorder showed faster Reaction Time (RT) for right than left hand conditions (reflecting the RH to LH transfer of information) whereas controls showed an opposite effect. Although the reading disabled group did not differ from controls on any of the RT measures of inter-hemispheric transfer time (see also Broman et al., 1985; Velay et al., 2002, cited in Badzakova-Trajkov et al., 2005), shorter transfer times under right hand conditions for both visual ($r=-.45$) and tactile ($r=-.63$) tasks was associated with poorer reading performance on the Gray oral word reading test among dyslexics. It was suggested that abnormally fast transfer of visual information from the RH to the LH affects processing in the LH.

Evidence for disrupted inter-hemispheric transfer time among dyslexics has also been found in several electrophysiological investigations (e.g., Davidson & Saron, 1992; Markee, Brown, Moore, & Theberge, 1996). For example, Davidson and Saron (1992) found that children with dyslexia showed faster inter-hemispheric transfer from RH to LH and slower inter-hemispheric transfer from LH to RH at occipital sites. However, Markee et al. (1996), using a more complex choice RT paradigm, found that adults with dyslexia were slower relative to controls in both directions at parietal sites. The findings of some other electrophysiological studies indicate that whereas normal readers showed greater sharing between hemispheres, dyslexics showed greater sharing within hemispheres (Leisman, 2002; Leisman & Ashkenazi, 1980).

A Left Hemisphere Linguistic Deficit

In addition to the research mentioned above, several studies have found gross anatomical and cytoarchitectonic abnormalities in the brains of deceased dyslexics that are consistent with a LH linguistic deficit (e.g., Galaburda & Kemper, 1979; Galaburda et al., 1985). These abnormalities have typically been found in LH perisylvian language areas, such as the superior temporal gyrus (including Wernicke's area) and the inferior premotor and prefrontal cortex (including Broca's area) (see Galaburda, 1999). Brain-imaging research implicates both ventral and dorsal posterior areas of the brain in the aetiology of dyslexia (for reviews see Pugh et al., 2001a; Pugh et al., 2001b; Salmelin & Helenius, 2004; Shaywitz, Lyon, & Shaywitz, 2006a).

The ventral (occipito-temporal) system is thought to constitute a fast memory based word identification system, whereas the dorsal (temporo-parietal) system, in conjunction with frontal areas (inferior frontal gyrus) is associated with coding and analysis of orthographic, phonological, and lexical-semantic characteristics of written words (for a review see Pugh et al., 2001a). As such, the dorsal system is thought to be important for basic decoding and analysis of

words and shows greater activation for low frequency and unfamiliar words. The ventral word recognition system develops with experience, becomes more active in skilled readers, and facilitates fluency in word recognition. The ventral system is thought to be partially dependent on the integrity of the dorsal system for normal reading development. An anterior (frontal) system, including the LH inferior frontal gyrus (BA 44), is thought to play a role in the articulatory recoding of print.

Dyslexics consistently show reduced activation in the ventral reading system (including lateral extra-striate and left occipito-temporal areas) during word and pseudoword reading (Brunswick et al., 1999; Helenius, Tarkiainen, Cornelissen, Hansen, & Salmelin, 1999b; Pugh et al., 2000; Rumsey et al., 1997b; Salmelin, Service, Kiesila, Uutela, & Salonen, 1996; Shaywitz et al., 1998). For example, using magnetoencephalography, it has been shown that normal readers, but not dyslexics, show letter string specific activation between 150-200ms post-stimulus in occipito-temporal areas (Helenius et al., 1999b; Salmelin et al., 1996). Similarly, using Positron Emission Tomography (PET), Brunswick et al. (1999) found a reduction in activation in the left posterior inferior temporal cortex (BA 37) among adult dyslexics during both explicit and implicit reading tasks, suggesting a specific impairment in lexical retrieval. A left occipito-temporal region known as the Visual Word Form Area (VWFA) is activated relatively automatically in response to word-like stimuli (including pseudowords) regardless of factors such as location, size, and font in normal readers (Cohen et al., 2000; Cohen et al., 2002; Dehaene, Le Clec'H, Poline, Le Bihan, & Cohen, 2002). Decreased activation in this area among those with dyslexia suggests difficulty in processing orthographic representations of words (Cao, Bitan, Chou, Burman, & Booth, 2006).

Dyslexics also show different patterns of activation in areas of the dorsal (temporo-parietal) reading system including the angular gyrus (BA 39), supramarginal gyrus (BA 40) and superior temporal cortex (Wernicke's area) (Cao et al., 2006; Flowers, Wood, & Naylor, 1991; Gross-Glenn et al., 1991; Helenius, Salmelin, Service, & Connolly, 1999a; Pugh et al., 2000;

Rumsey et al., 1997b; Salmelin et al., 1996; Shaywitz et al., 1998; Simos, Breier, Fletcher, Bergman, & Papanicolaou, 2000). This dorsal system is thought to be important for analysing and learning the relationships between orthography and phonology (Pugh et al., 2001b). In particular, dyslexics typically show reduced activation in the angular gyrus which is thought to be important for the pre-lexical processing of letter strings and the cross-modal integration of information required during phonological and semantic coding of words (Pugh et al., 2000; Rumsey et al., 1997b; Salmelin et al., 1996). A reduction in activation in the middle temporal gyrus has been purported to reflect ineffective use of semantic representations (Cao et al., 2006).

Reduced activation in LH posterior areas among dyslexics is often coupled with increased 'compensatory' activation of inferior frontal and right hemisphere posterior regions (Brunswick et al., 1999; Pugh et al., 2000; Richards et al., 1999; Salmelin et al., 1996; Shaywitz et al., 1998; Simos et al., 2000). For example, Shaywitz et al. (1998) found greater activation in RH temporo-parietal areas and the inferior frontal gyrus in dyslexia as a function of increased phonological demands. Furthermore, activation of the angular gyrus and middle temporal gyrus was greater in the LH for controls and in the RH for dyslexics. Other studies have reported correlations between reading performance and right hemisphere temporo-parietal activation for dyslexic but not normal readers (e.g., Rumsey et al., 1999). Compensatory activity in the RH is thought to relate to the development of a visuo-semantic pattern recognition system that is not phonologically based (Pugh et al., 2001a). Although several studies have shown increased compensatory activation in the left inferior frontal gyrus (Brunswick et al., 1999; Shaywitz et al., 1998; Temple et al., 2001), others have failed to find differences (Paulesu et al., 1996; Rumsey et al., 1997b; Shaywitz et al., 2003), or have reported decreased activation in dyslexia (Georgiewa et al., 1999). In general these findings have indicated that whereas compensated adult dyslexics often show greater activation, children with dyslexia often show under activation in this area (Cao et al., 2006). Compensatory activity in frontal areas is thought to relate to an increased reliance on articulatory coding during reading (Pugh et al., 2001a) and/or a top-down

compensatory process of matching words against a mental template (Salmelin et al., 1996). The underactivation observed among dyslexic children is thought to reflect less top down modulation of orthographic and phonological representation in posterior regions (Cao et al., 2006).

In a functional MRI study, activation of these reading systems were investigated in compensated adult dyslexics, persistently impaired adult readers, and non impaired controls (Shaywitz et al., 2003). During a pseudoword rhyme decision task, both poor reading groups showed a reduction in activation in superior temporal and occipito-temporal regions, and greater activation in the right inferior frontal gyrus. Compensated dyslexic adults showed additional compensatory activation in RH superior frontal and middle temporal gyri and the LH anterior cingulate gyrus. However, during a real word semantic decision task, compensated dyslexics showed a reduction in activation of LH posterior regions relative to controls and persistently impaired readers showed greater activation relative to controls in the occipito-temporal visual word form area. Furthermore, whereas normal readers showed connectivity between the visual word form area and the left inferior temporal gyrus, persistently impaired readers showed connectivity between the visual word form area and RH prefrontal areas. These findings indicated that the visual word form area functions as part of the memory network in persistently poor readers and it was suggested that they are more likely to use rote memory to recognise words. Taken together these findings are consistent with the distinction between a genetic type of dyslexia with high IQ scores (compensated dyslexics) and a more environmentally influenced type of dyslexia with relatively low IQs (persistently impaired readers) (see Shaywitz et al., 2003). In a recent intervention study, an experimental phonology based treatment was found to produce changes in reading accuracy, fluency, and comprehension as well as patterns of brain activation. Findings indicated that the intervention lead to the development of neural systems in both anterior (inferior frontal gyrus) and posterior (middle temporal gyrus) areas of the brain (Shaywitz et al., 2006a). In another intervention study, dyslexic children who showed normalising changes (increased left temporo-parietal activity) as a result of an intervention

showed greater reading improvements than those who showed compensatory changes (increased right temporo-parietal and frontal activity) in activation during the performance of decoding tasks (Simos et al., 2007).

There is also some evidence that dyslexia is associated with less functional connectivity between language areas (Horowitz, Rumsey, & Donohue, 1998; Paulesu et al., 1996; Pugh et al., 2000). For example, Horowitz et al. (1998) found lower correlations between the LH angular gyrus and other occipital and temporal sites among dyslexics. Consistent with the phonological deficit hypothesis, Pugh et al. (2000) found that the functional connectivity between dorsal (angular gyrus) and ventral (occipito-temporal) areas was disrupted in dyslexia for tasks requiring orthographic to phonological assembly. Further, consistent with compensatory processing, dyslexics showed greater functional connectivity in homologous areas of the RH relative to controls. The dorsal and ventral visual processing streams are thought to underlie sublexical and lexical (whole word) processing of words respectively (Borowsky et al., 2006; Posner & Raichle, 1994).

Paulesu et al. (1996) found that adult compensated dyslexics with phonological processing impairments showed a disrupted connection between the inferior parietal cortex (including Wernicke's area) and the anterior system (including Broca's area). These areas were activated independently by short-term memory (unsegmented phonology) and rhyming (segmented phonology) tasks respectively, but were not activated concurrently, suggesting segregation between these two systems involved in phonological coding. The lack of activation of the left insula in dyslexia was thought to underlie the disrupted connectivity between these two areas. Further, increased activation of the insula has been observed among normal readers for phonological relative to orthographic tasks (Rumsey et al., 1997a) and the insula cortex has been shown to be sensitive to phonology (Borowsky et al., 2006). Consistent with the proposal of a disconnection syndrome, in a recent MRI study it was found that adults with dyslexia

showed less development of white matter in the LH in comparison to controls, suggestive of a reduction in myelination (Klingberg et al., 2000).

The involvement of LH linguistic areas in developmental dyslexia is also supported by findings of acquired dyslexia or alexia following damage to LH areas. Alexias have been categorised into posterior alexia (occipital alexia or alexia without agraphia), central alexia (parietotemporal alexia or alexia with agraphia) and anterior alexia (frontal or motor alexia associated with Broca's aphasia) (Benson, 1977). Posterior alexia has been associated with a number of anatomical areas including the LH angular gyrus, the medial occipital lobes, Broca's area, and the splenium of the corpus callosum (Damasio & Damasio, 1983). Anatomical lesions to areas of the LH also result in acquired forms of dysphonesia (supramarginal gyrus and insula), and dyseidesia (left posterosuperior angular gyrus and parietooccipital lobule) (Roeltgen & Heilman, 1984). It has also been suggested that surface and phonological alexia occur as a result of lesions to the left posterior temporal lobe and left temporo-parietal areas respectively (McCarthy & Warrington, 1990).

Although activation in left occipito-temporal areas has been shown to correlate positively with reading skill in children (Shaywitz et al., 2002) and some studies have shown increased activation of LH areas following phonological interventions (see Shaywitz, Mody, & Shaywitz, 2006b; Simos et al., 2007; Vellutino et al., 2004), it is possible that reading specific areas fail to develop due to neurological problems in other areas. For example, brain abnormalities such as ectopias develop prior to the completion of neuronal migration to cortical as well as subcortical areas such as the thalamus. Alteration in neuronal size and cellular asymmetry has also been observed in the primary visual cortex, such that normal readers but not dyslexics showed larger neurons in the LH in comparison to the RH (Jenner, Rosen, & Galaburda, 1999). Thus it is possible that sensory/perceptual abnormalities cause secondary linguistic/cognitive abnormalities and vice versa (Galaburda, 1999). Furthermore, learning to

read and write can also change brain organisation, and in turn modify the LH dominance for language (Ostrosky-Solis, Garcia, & Perez, 2004).

The Cerebellar hypothesis

According to the cerebellar theory of dyslexia, a cerebellum impairment causes the phonological core deficit in dyslexia due to its role in the development of articulatory skills that are important for subsequent language development and in particular phonemic awareness (for reviews see Nicolson & Fawcett, 1999; Nicolson, Fawcett, & Dean, 2001). A cerebellar deficit is also proposed to account for slowed central processing speed including rapid automatised naming deficits, and difficulties in spelling and writing. This hypothesis stemmed from the observation that dyslexics are impaired on a range of skills (balance, motor skill, phonological skill, working memory, information processing speed) associated with skill automatization (Nicolson & Fawcett, 1999). Children with dyslexia also show poorer performance than both chronologically age matched and reading age matched controls on tasks assessing the clinical symptoms (e.g., muscle hypotonia, ataxia) of cerebellar dysfunction (Fawcett, Nicolson, & Dean, 1996; Fawcett et al., 2001). Furthermore, Nicolson, Fawcett, and Dean (1995) found the same dissociation between time and loudness estimation in dyslexia that is found in patients with cerebellar damage. There is also evidence from brain imaging research for cerebellar abnormalities (particularly in the right cerebellum) in dyslexia. For example, Rae et al. (2002) observed rightward asymmetry of the cerebellum in controls, and an association between lack of asymmetry and phonological decoding (nonword reading) in dyslexics. The right cerebellum predominantly links to the left hemisphere as well as pre-motor and pre-frontal areas including Broca's area, and is activated during silent word generation and verbal working memory tasks. Dyslexics also show decreased activation in the right cerebellum during both learning and execution of a motor sequence, coupled with decreased activation in the left anterior cingulate during the execution phase (Nicolson et al., 1999). However, during the performance of an

auditory word repetition task, adult dyslexics show reduced PET activation in the left cerebellum (McCrory, Frith, Brunswick, & Price, 2000).

Although there is clear evidence for cerebellar involvement in dyslexia, the causal relationship between cerebellar problems and reading disability has been debated in the literature (e.g., Ivry & Justus, 2001; Ramus et al., 2003; Zeffiro & Eden, 2001). Some studies have failed to show skill automatization deficits (Wimmer, Mayringer, & Landerl, 1998) or have found a skill automatization deficit only among dyslexics with high Attention Deficit Hyperactivity Disorder (ADHD) symptoms (Wimmer, Mayringer, & Raberger, 1999). Ramus et al. (2003) found that only four out of 16 dyslexics showed evidence for a cerebellar deficit. Further, dyslexics do not show the extent of symptoms exhibited by patients with acquired cerebellar disorders, and those with cerebellar disorders do not typically show marked reading or phonological problems (Ivry & Justus, 2001; Zeffiro & Eden, 2001) (but see Scott et al., 2001 for evidence of language deficits following developmental cerebellar lesions). It has been argued that it is impairment in other areas of the brain (e.g., perisylvian language areas) that affect the functioning of the cerebellum in dyslexia (Zeffiro & Eden, 2001), and that cerebellar impairment is a correlate rather than a cause of reading disorders (Ivry & Justus, 2001). A recent conceptualisation (discussed later in this chapter) suggests that cerebellar impairments occur due to an underlying temporal processing or magnocellular deficit that affects more than one sensory modality.

The Auditory Deficit Hypothesis

According to the auditory deficit hypothesis, an inability to discriminate the temporal order of rapid auditory stimuli underlies the phonological deficits observed in dyslexia (for reviews see Farmer & Klein, 1995; Tallal, Miller, & Fitch, 1993; Tallal, Miller, Jenkins, & Merzenich, 1997; Vellutino et al., 2004). Despite normal performance on auditory tasks involving single stimuli, dyslexics perform below normal readers on auditory temporal order

judgement tasks, particularly when short inter-stimulus intervals are used (Tallal, 1980, 1984) (but see Share, Jorm, Maclean, & Matthews, 2002). Significant correlations have also been reported between performance on these tasks and both phonological ability (Farmer & Klein, 1993; Tallal, 1980) and nonword reading ability (Cestnick & Jerger, 2000). It has been proposed that a deficit in temporal processing of rapidly changing auditory stimuli affects the perception of speech signals and the development of phonemic awareness in dyslexics and therefore causes a phonological deficit. Based on findings that dyslexics also take longer to make temporal order judgments in the visual modality and are impaired on other visual tasks that require rapid temporal processing (Lovegrove, 1996), it has been further proposed that dyslexics suffer from a generalised temporal processing deficit regardless of stimulus modality (Farmer & Klein, 1995; Tallal et al., 1993). This hypothesis is considered further later on in this chapter.

There is no doubt that dyslexia is associated with auditory processing differences, however much of the evidence is correlational and based on language impaired rather than dyslexic subjects. Although Tallal has demonstrated language improvements following training designed to reduce temporal integration thresholds in language impaired individuals (Tallal et al., 1997), the causal nature of this hypothesis is questionable in relation to dyslexia (see Mody, Studdert-Kennedy, & Brady, 1997; Snowling, 2001; Studdert-Kennedy, 2002; Studdert-Kennedy & Mody, 1995). In addition, the auditory deficit theory is unable to account for deficits in orthographic processing (e.g., developmental surface dyslexia, dyslexia), and fails to explain either why dyslexics are able to speak and comprehend language fluently or why some dyslexics have phonological deficits without auditory processing deficits (Ramus et al., 2003). Dyslexics with an auditory impairment also typically have problems with speech perception indicating that auditory deficits are a correlate rather than a cause of dyslexia (see Hogben, 1996; Ramus, 2004). For example, Tallal and Stark (1982) found no differences in performance on temporal order judgment tasks in dyslexics without a concomitant oral (receptive and expressive) language delay. However, these dyslexics did not differ from controls in terms of

their nonword reading ability either. In a recent longitudinal study, early temporal deficits did not predict later phonological impairment, pseudoword reading, or dyslexia, but did predict oral receptive vocabulary and reading comprehension problems (Share et al., 2002). It has also been argued that the performance of dyslexics on auditory temporal order judgment tasks occurs due to a deficit in speech discrimination rather than a rate of auditory processing deficit (Mody et al., 1997; Studdert-Kennedy & Mody, 1995) (for recent reviews see Klein, 2002; Vellutino et al., 2004).

The Magnocellular Hypothesis

Several lines of converging evidence provide support for the hypothesis that dyslexia stems from a specific low level deficit in the magnocellular visual processing pathway (for reviews see Greatrex & Drasdo, 1995; Lovegrove, 1996; Lovegrove, Martin, & Slaghuys, 1986b; Lovegrove, Garzia, & Nicholson, 1990; Stein, 2001a; Stein & Talcott, 1999; Stein & Walsh, 1997). The sustained/parvocellular and transient/magnocellular pathways are two parallel and complimentary sub-systems in the human visual system that have relatively distinct anatomical projections and functional characteristics (for reviews see Breitmeyer & Ganz, 1976; Enroth-Cugall & Robson, 1966; Livingstone & Hubel, 1987; Merigan & Maunsell, 1990; Milner & Goodale, 1995; Shapley & Perry, 1986; Zeki, 1992). The parvocellular (P) system projects via the dorsal Lateral Geniculate Nucleus (LGN) of the thalamus, to layers IV β of the primary visual cortex (Area V1), and then mainly to the infero-temporal cortex of the temporal lobe. The faster magnocellular (M) system predominates in peripheral vision and is composed of large, thickly myelinated cells that allow fast conduction. Their larger size and high convergence promotes greater spatial summation and sensitivity to light over a large area. The M pathway projects from the retina via the ventral lateral geniculate nucleus of the thalamus to layer IV α of the primary visual cortex, and then predominantly to the PPC, as well as the frontal eye fields, superior colliculus, and cerebellum (see Livingstone & Hubel, 1987).

Perhaps the most compelling evidence for an M deficit in dyslexia are the abnormalities observed in the magnocellular layers of the lateral geniculate nucleus of the thalamus in the brains of deceased dyslexics (Livingstone et al., 1991). However, the link between M functioning and dyslexia first came from psychophysical studies showing a reduction in contrast sensitivity to sinusoidal gratings, increased duration of visible persistence and abnormal masking functions among dyslexics (see Lovegrove, 1996; Lovegrove et al., 1986b). The sustained and transient systems are most sensitive to high spatial/low temporal frequencies and low spatial/high temporal frequencies respectively and dyslexics show reduction in contrast sensitivity to low relative to high spatial frequency static grating stimuli (Lovegrove et al., 1982; Martin & Lovegrove, 1984, 1988) and to flickering gratings at a range of temporal frequencies (Brannan & Williams, 1988; Cornellissen, 1993; Felmingham & Jakobson, 1995; Martin & Lovegrove, 1987, 1988; Mason, Cornelissen, Fowler, & Stein, 1993), particularly at high temporal frequencies (Ben-Yehudah, Sackett, Malchi-Ginzberg, & Ahissar, 2001; Felmingham & Jakobson, 1995; Martin & Lovegrove, 1987). Some studies, however, have failed to find any significant effects (Gross-Glenn et al., 1995; Hayduk, Bruck, & Cavanagh, 1996), or have found effects that are not consistent with the theoretical predictions (Olson & Datta, 2002).

Visible persistence or the brief visible trace of an image after its physical offset is thought to reflect the sustained activity of the P system. Studies that have employed gap detection tasks have found that dyslexics show a longer duration of visible persistence relative to normal readers (Badcock & Lovegrove, 1981; Di Lollo, Hansen, & McIntyre, 1983; Lovegrove, Heddle, & Slaghuis, 1980; Martos & Marmolejo, 1993; Slaghuis & Lovegrove, 1984; Slaghuis & Lovegrove, 1985; Slaghuis, Lovegrove, & Davidson, 1993). For example, dyslexics show a smaller increase in visible persistence as a function of spatial frequency, suggestive of a reduction in transient (M) on sustained (P) inhibition (Lovegrove et al., 1980; Slaghuis & Lovegrove, 1985). However, some studies have failed to replicate findings of increased visible persistence in dyslexia, particularly those that have used temporal integration tasks to measure

visible persistence (Di Lollo et al., 1983; Hogben, Roding, Clark, & Pratt, 1995).

Other studies have measured visible persistence and M and P interactions indirectly using visual masking paradigms (Di Lollo et al., 1983; Slaghuis, Lovegrove, & Freestun, 1992; Slaghuis & Pinkus, 1993; Stanley & Hall, 1973; Williams & LeCluyse, 1990; Williams, LeCluyse, & Bologna, 1990; Williams, Molinet, & Le Cluyse, 1989) and Ternus apparent motion tasks (Cestnick & Coltheart, 1999; Davis, Castles, McAnally, & Gray, 2001; Slaghuis & Ryan, 1999; Slaghuis, Twell, & Kingston, 1996). A common finding in the masking literature is that masking occurs at longer inter-stimulus intervals among dyslexics relative to controls (Slaghuis et al., 1992; Slaghuis & Pinkus, 1993; Williams et al., 1989) with some studies showing an attenuation of masking in the periphery (Williams & LeCluyse, 1990; Williams et al., 1989). Similarly, dyslexics perceive Ternus group movement at shorter inter-stimulus intervals relative to normal readers (Cestnick & Coltheart, 1999; Davis et al., 2001) and less group movement at longer inter-stimulus intervals (Cestnick & Coltheart, 1999; Slaghuis et al., 1996). Initially the findings of greater visible persistence and differing masking and apparent motion functions in dyslexia were attributed to a lack of inhibition of the sustained (P) system by the transient (M) system (see Lovegrove et al., 1986b). This lack of ‘transient on sustained inhibition’ was argued to affect saccadic suppression in dyslexia resulting in retinal image blur and visual instability. However, this hypothesis has since been challenged and is discussed in more detail below.

Transient deficits have been shown to precede commencement of reading and continue into adulthood indicating that they do not occur as a result of reading disability (Slaghuis & Pinkus, 1993; Slaghuis et al., 1996). Using combined data from previous studies, Lovegrove et al. (1986) reported that approximately 75% of dyslexics could be classified as showing a transient system deficit based on the visible persistence vs. spatial frequency regression slope. Studies have also shown that the addition of a uniform flickering field, thought to selectively engage the transient system, results in a reduction in the difference between dyslexics and

controls on tasks measuring contrast sensitivity (Martin & Lovegrove, 1988), visible persistence (Slaghuis & Lovegrove, 1984), and Ternus apparent motion (Slaghuis et al., 1996). It has been suggested that flicker masking decreases M activity in controls, but has little effect on dyslexics who already suffer from a sluggish transient system (Slaghuis & Lovegrove, 1984). More recently, lower sensitivity to the frequency doubling illusion (thought to be mediated solely by M cells) has been shown in both dyslexic children (Pammer & Wheatley, 2001), and adults (Buchholz & McKone, 2004), coupled with normal performance on a task assessing parvocellular function. Several evoked potential studies have found electrophysiological evidence for a magnocellular deficit in dyslexia (Lehmkuhle, Garzia, Turner, Hash, & Baro, 1993; Livingstone et al., 1991; May, Lovegrove, Martin, & Nelson, 1991). These studies are discussed in more detail in Chapter 4.

There is also some evidence for a relationship between magnocellular functioning and phonological ability. For example, studies employing the Boder classification system have reported evidence for an M deficit in dysphonetic or severe dysphonetic dyslexics but not dyseidetic dyslexics (Borsting et al., 1996; Ridder, Borsting, Cooper, McNeel, & Huang, 1997; Slaghuis & Ryan, 1999). No previous study has reported differences between dyseidetic (surface) dyslexics and controls on measures of contrast sensitivity (Borsting et al., 1996; Ridder et al., 1997; Slaghuis & Ryan, 1999), and one study found a reduction in sensitivity to high spatial frequencies among dyseidetic dyslexics (Spinelli et al., 1997).

Among unselected children, Lovegrove, Bowling, Slaghuis, Geeves, & Nelson (1986a) reported a positive correlation ($r=0.34$) between contrast sensitivity at age 6 and reading ability at age 8 after controlling for IQ. In another study, measures of phonological recoding and phonological awareness were found to load onto the same factor as flicker sensitivity measures in a group of dyslexics and controls (Lovegrove, McNicol, Martin, Mackenzie, & Pepper, 1989). Slaghuis et al. (1996) did not find a significant correlation between non-word reading and performance on the Ternus task (possibly due to ceiling effects in nonword reading scores of

controls), but a discriminant analysis showed that both measures in combination correctly classified all participants into either the dyslexic or control group. Cestnick and Coltheart (1999) found that phonological dyslexics differed from surface dyslexics and controls on the Ternus task and reported a significant relationship between nonword reading and performance on the Ternus task after accounting for irregular word reading. Similarly, Davis et al. (2001) found that performance on the Ternus task was more strongly related to nonword than irregular word reading, after controlling for the effects of inattention. Together these findings indicate a relationship between poor phonological skills and magnocellular or transient system functioning. However, many of these studies did not include a measure of orthographic processing (such as irregular word reading). Furthermore, in a recent study in which dyslexic children were classified on the basis of irregular word and nonword reading, there were no differences in contrast sensitivity for stimuli designed to maximally stimulate either the magnocellular or parvocellular systems (Williams, Stuart, Castles, & McAnally, 2003).

Despite converging evidence for a low level transient deficit in dyslexia from a number of different laboratories, this hypothesis has been the subject of criticism in the literature (Skottun, 2000, 2001a, 2001b; Skottun & Parke, 1999; Stuart, McAnally, & Castles, 2001). It has been argued that only a few contrast sensitivity studies provide evidence for a selective M deficit and that these studies are outnumbered by those that have failed to find any significant effects or have found effects that are inconsistent with theoretical predictions (see Skottun, 2000). The use of metacontrast and apparent motion paradigms to measure M functioning has also been criticised (Skottun, 2001a, 2001b).

It has also been argued that general inattention or motivation differences could account for the psychophysical functions observed in many experiments (Davis et al., 2001; Stuart et al., 2001). However, this claim is not consistent with experiments finding no group differences on psychophysical tasks that measure P system functioning (Lovegrove et al., 1986b; Lovegrove et al., 1990), or those that have shown increased sensitivity at high spatial frequencies or measured

vigilance by including catch trials (see Stein, 2003). Further, Davis et al. (2001) found that that differences in performance on the Ternus task remained after controlling for the effects of inattention.

It is also possible that the inconsistent findings in the literature occurred due to factors such as subject selection and sensitivity of the measures being used (see Hogben, 1996; Stein, 2003; Stein, Talcott, & Walsh, 2000). The dyslexic participants from the Lovegrove laboratory were all males selected according to reading accuracy with most showing a concurrent deficit in nonword reading (though irregular word reading was not necessarily assessed). In contrast, the selection criteria used in other studies has varied and is often based on referral from special clinics or schools which may produce diagnostic bias (Hogben, 1996).

It has been argued that the visual problems experienced in dyslexia are a neurobiological correlate rather than a cause (Eden & Zeffiro, 1998). Thus, the largest challenge for proponents of the M hypothesis has been to explain how an M deficit causes the reading problems experienced by dyslexics. For example, the proposal that a lack of transient on sustained inhibition results in abnormal saccadic suppression has been challenged by research indicating that the transient rather than the sustained system is inhibited during saccades (Burr, Morrone, & Ross, 1994). Another possible way that a transient deficit may affect reading is through a lack of integration across fixations due to poor utilisation of information in the periphery (see Lovegrove et al., 1986b; Lovegrove et al., 1990). For example, transient channels are thought to play a role in extracting information from peripheral vision during fixations in order to enhance visual recognition during subsequent fixations (Breitmeyer, 1980). This proposal is consistent with findings that dyslexics make more errors when reading continuous text than isolated words (Lovegrove, 1996), but does not account for the single word recognition deficits typically observed in dyslexia and the fact that visual confusions usually involve neighbouring letters rather than words separated by saccades (see Stein & Walsh, 1997). Another possible explanation is that an M deficit magnifies to produce deficits in extra-striate visual areas that

receive predominantly M input and are involved in motion perception, eye movements, and spatial attention (Breitmeyer, 1989; Lovegrove et al., 1986b). Stein and colleagues have expanded this hypothesis further in recent years and have argued that an M deficit magnifies to produce deficits in functions mediated by the PPC (see Stein & Walsh, 1997).

The Posterior Parietal Cortex

The P and M visual pathways have also been termed the ‘What’ and ‘Where’ or ‘ventral’ and ‘dorsal’ visual pathways respectively as each projects preferentially to functionally specialised extra-striate visual areas. As previously mentioned, the P system projects preferentially to the infero-temporal cortex whereas the M system projects preferentially to the PPC and both of these pathways pass through different extra-striate areas of the visual system. For example, some extra-striate areas receive predominantly M input and are involved in the processing of motion (V5/MT) and dynamic form (V3), while other areas receive predominantly P input and are involved in processing colour (V4) and form (V2) (Schiller, 1996; Zeki, 1992). Although these pathways are relatively anatomically and functionally distinct they also interact considerably with some intermingling of input to extra-striate areas and many reciprocal connections between these areas (Schiller, 1996). However, the ‘what’ pathway is primarily responsible for the resolution of fine detail, colour, and the analysis and recognition of form, whereas the ‘where’ pathway is involved in motion sensitivity, normal eye movement control, the visual guidance of action, and visuospatial attention (Milner & Goodale, 1995). The M-system also projects to other areas that are important for visuo-motor control and spatial attention including the frontal eye fields, cerebellum, and superior colliculus of the thalamus (Milner & Goodale, 1995). Thus, during reading, the M system is important for locating the position, spatial relationships, and movement of words/letters, whereas the parvocellular system is important for determining information about colour, pattern, and the identity of words/letters (Castles & Coltheart, 1993; Slaghuys et al., 1996). Based on findings of deficits in motion

sensitivity, eye movements, peripheral vision, and visual attention, it has been argued that dyslexia is associated with a specific abnormality of the PPC (Stein, 2003; Stein & Walsh, 1997). Further, considering the role of the RH PPC in visuo-spatial function, a RH posterior deficit has often been suggested to underlie the difficulties observed in dyslexia.

Motion Sensitivity

Area MT in the superior temporal sulcus is sensitive to the direction of motion, is activated during motion discrimination tasks in the Macaque monkey (Newsome & Pare, 1988), and is mediated by magnocellular input from the lateral geniculate nucleus of the thalamus (Maunsell, Nealey, & DePriest, 1990). In humans, the analogous Area MT/V5 plays a role in direction discrimination, detecting of shearing motion, detection of 3D structure from motion, and the guidance of eye movements to moving targets (Eden et al., 1996). As well as receiving magnocellular input, Area MT also receives input from the non-geniculo-striate areas such as the superior colliculus and pulvinar of the thalamus (Eden et al., 1996). Studies investigating motion sensitivity have shown that both children and adults with dyslexia produce higher detection thresholds or lower motion sensitivity in comparison to control groups (Cornelissen, Richardson, Mason, Fowler, & Stein, 1995; Everatt, Bradshaw, & Hibbard, 1999a; Hansen, Stein, Orde, Winter, & Talcott, 2001; Raymond & Sorenson, 1998; Richardson, 1995; Ridder, Borsting, & Banton, 2001; Talcott, Hansen, Assoku, & Stein, 2000a; Witton et al., 1998). Similarly, brain imaging studies have demonstrated elevated speed discrimination thresholds in dyslexia coupled with reduced activation in Area MT relative to controls (Demb, Boynton, Best, & Heeger, 1998a; Demb, Boynton, & Heeger, 1997, 1998b; Eden et al., 1996). Eden and Zeffiro (1998) suggest that these neuroimaging findings coupled with those that have investigated left hemisphere linguistic processing (e.g., Shaywitz et al., 2003) indicate a common anatomical basis related to functioning of inferior parietal areas.

The results of some motion sensitivity studies are consistent with the proposal that

dyslexia is associated with abnormal MT or PPC function rather than a low level M deficit. For example, Raymond and Sorenson (1998) found that group differences increased as a function of the number of frames, suggesting poor perceptual integration or temporal recruitment, rather than poor low level motion detection *per se*. Similarly, Hill and Raymond (2002) found that dyslexics needed a greater number of frames to detect movement in transparent bidirectional displays associated with the involvement of Area MT and not Area V1 of the visual system. However, functional MRI studies have revealed conflicting findings. One study found that adult males with dyslexia showed reduced activation in Area MT but similar activation in Areas V1/V2 relative to controls during speed discrimination tasks (Eden et al., 1996) and another study showed reduced activity in both Area V1 and Area MT (Demb et al., 1998b). Furthermore, another study showed similar activation of Area MT in adults with dyslexia and controls in response to high contrast apparent motion stimuli (Vanni, Uusitalo, Kiesila, & Hari, 1997).

Some studies have reported a link between coherent motion sensitivity and letter position encoding in groups of unselected children and adults that are suggestive of a relationship between motion sensitivity and orthographic skill (Cornelissen & Hansen, 1998; Cornelissen, Hansen, Hutton, Evangelinou, & Stein, 1998; Talcott et al., 2002; Talcott et al., 2000b). Together these findings suggest that reading errors are caused by uncertainty about the positions of letters and features with respect to each other. For example, Talcott et al. (2000b) found that motion sensitivity correlated more strongly with irregular word reading and a pseudo-homophone test (requiring orthographic skill) than with tasks requiring phonological skills (nonword reading and spoonerisms) in a group of unselected children. Furthermore, motion sensitivity accounted for unique variance in orthographic skill after phonological skill was accounted for. Similarly, a positive but non-linear relationship was found between coherent motion thresholds and the probability of making letter errors (orthographically inconsistent reading errors) in a group of 60 unselected children (Cornelissen et al., 1998), with no correlation observed between coherent motion thresholds and performance on phonological

measures (spoonerisms, rhyme detection).

Despite evidence for an association between motion sensitivity and orthographic processing, other findings are more consistent with a relationship with phonological processing. For example, Slaghuis and Ryan (1999) found that only dysphoneidetics showed a significant reduction in motion sensitivity relative to controls. However, the findings of another study indicated that lower motion sensitivity was characteristic of all Boder subtypes (Ridder et al., 2001). Talcott et al. (1998) found that coherent motion thresholds and critical fusion frequencies (flicker sensitivity) together correctly classified 77.8% of adult dyslexics ($n=18$) and controls ($n=18$) and accounted for nearly 48% of the variance in nonword reading. Furthermore, whereas both visual measures correlated moderately with nonword reading, there was a stronger correlation between the combined visual processing score and nonword reading ($r=0.691$). Witton et al. (1998) also reported a correlation between nonword error time (speed and accuracy) and visual motion sensitivity ($r=.406$). However, a measure of orthographic processing was not included in either of these studies, and Witton et al. found a stronger correlation between nonword error time and sensitivity to low frequency (2Hz) dynamic auditory stimuli ($r=.603$) which is discussed further below. Interestingly Area V5 is also activated during a range of phonological tasks (e.g., phoneme deletion, pig Latin conversion) in both the auditory and visual modality suggesting that this area may be involved in fast transient processing in both modalities (Liederman et al., 2003). Further, Leiderman et al. (2003) found that repetitive transcranial magnetic stimulation to Area V5 disrupted nonword reading but not performance on phonological and orthographic decision tasks. It was concluded that Area V5 may not be involved in phonological decoding *per se* but may be important for image stabilisation and/or letter localisation.

One criticism of studies investigating motion sensitivity is the fact that the motion sensitivity scores of dyslexics and controls often overlap and greater variability is observed in dyslexic groups due to outliers (Cornelissen et al., 1995; Everatt et al., 1999a; Talcott et al.,

1998). As previously mentioned, it has also been argued that visual processing deficits contribute to reading ability independently of phonological processes (Valdois et al., 2004) and that M deficits are a correlate or a biological marker rather than a cause of developmental dyslexia (Vellutino et al., 2004). One recent study identified a subgroup of six (out of 30) adult dyslexics with a nonword reading deficit who were impaired across a range of magnocellular tasks (flicker detection, detection of drifting low spatial frequency drifting gratings, speed discrimination thresholds, coherent motion sensitivity) as well as other perceptual tasks (Amitay, Ben-Yehudah, Banai, & Ahissar, 2002). The performance of the remainder of the dyslexics did not differ from controls on the magnocellular tasks, but did differ from controls on visual and auditory tasks requiring fine discriminations.

In summary, while decreased motion sensitivity is common in dyslexia, it is likely that this only applies to some groups of dyslexics. Although some research findings suggest a link between motion sensitivity and nonword reading or phonological processing in dyslexia, motion sensitivity deficits are more likely to be related to orthographic processing in groups of unselected children. Further research is required to investigate the relationship between component reading skills and motion sensitivity deficits among subgroups of dyslexics.

Eye movements and Binocular control

Whereas normal eye movement control has been observed in dyslexia using standard clinical tests, other studies using more sensitive tests have found evidence for binocular instability, impaired accommodation, and eye movement control (for reviews see Stein, 2001a; Stein & Talcott, 1999). In particular, binocular vergence control is often poor in children with dyslexia and they are unable to maintain stable and accurate fixation (Eden, Stein, Wood, & Wood, 1994; Evans, Drasdo, & Richards, 1994; Fowler & Stein, 1979; Stein & Fowler, 1980). It has been argued that unstable eye control in dyslexia leads to unstable visual perceptions and visual reading errors (Stein, 2001a; Stein & Talcott, 1999). This is consistent with anecdotal

reports that letters “appear to move around, to change places, to merge with each other, to move in or out of the page, to blur or suddenly get larger or smaller” (Stein, 2001b, p. 22) and may also explain letter reversal and anagram errors made during reading. However, the use of the Dunlop test to measure ocular dominance has been criticised as the findings of some investigators have failed to support those of Stein and Fowler (e.g., Newman et al., 1985; Bishop et al., 1979, cited in Mason et al., 1993). Furthermore, Moores et al. (1998) found no evidence for differences in vergence control across saccades in adults with dyslexia. It has also been argued that eye movement deficits result from an underlying linguistic deficit (Morris & Rayner, 1991).

Stein has argued that the relationship between binocular control and reading ability is causal in nature as the same difficulties are not present in reading age matched controls (see Stein, 1989). Further, monocular occlusion therapy has been shown to improve both binocular stability and reading performance (Cornelissen, Bradley, Fowler, & Stein, 1992; Stein, Richardson, & Fowler, 2000). Monocular occlusion is thought to improve the *utrocular control* that is essential for fixating accurately and steadily on visual targets (Stein, 2001a). It has been suggested that poor binocular control affects orthographic rather than phonological processing. This is supported by evidence that children with binocular instability make more visual nonword reading errors and more phonologically plausible spelling errors (particularly when reading irregular words) suggesting a propensity to spell and read phonetically (Cornelissen, Bradley, Fowler, & Stein, 1991; Cornelissen, Bradley, Fowler, & Stein, 1994). Furthermore, an index of binocular fixation has been shown to contribute unique variance to reading scores after accounting for the contribution of age, IQ, and phonological recoding (pig Latin completion time) (Eden, Stein, & Wood, 1993). Thus unsteady fixation and poor vergence control may determine the ability to discriminate the correct order of letters and therefore disrupt the learning and memory of the orthographic rules of language (Cornelissen et al., 1991; Cornelissen et al., 1994).

The PPC and the superior colliculus are both integral in controlling eye movements and the voluntary visual guidance of other movements and the M system is thought to be important for both stabilising fixations and directing eye movements between fixations (Stein, 2003). In a study investigating both ocular dominance (Dunlop test) and flicker contrast sensitivity, it was found that dyslexics who failed the Dunlop test were significantly less sensitive to flickering gratings compared to those who passed, suggesting a link between flicker sensitivity and ocular dominance (Mason et al., 1993). Evans et al. (1994) found reduced visual acuity, binocular instability (reduced vergence amplitude and stability), reduced amplitude of accommodation, reduced contrast sensitivity at low spatial frequencies, and reduced sensitivity to uniform flicker in a sample of dyslexic children ($n=39$) in comparison to control children ($n=43$). Measures of contrast sensitivity at low spatial frequencies and of flicker sensitivity were not correlated. However, flicker sensitivity and low vergence amplitudes were correlated in dyslexia, suggesting a link between sensory-motor and visual deficits.

Poor binocular control and impaired ability to fixate on small targets is likely to lead to less accurate spatial localisation of visual images. This is consistent with other research showing that dyslexics are less accurate at localising visual stimuli (Graves, Frerichs, & Cook, 1999; Riddell, Fowler, & Stein, 1990; Solman & May, 1990). For example, Solman and May (1990) found that poor readers are less able to localise briefly presented letters or shapes, particularly those presented further from fixation. Further, dyslexics with unstable binocular control are poorer than controls at localising small dots, particularly in the LVF (Riddell et al., 1990). It was suggested that these children have trouble with maintaining an accurate spatial map.

Together these findings implicate the functioning of the RH in developmental dyslexia. Patients with RH parietal lesions also experience poor fixation, poor vergence control, and eye movement abnormalities, particularly for stimuli presented in the LVF (Fowler, Richardson, & Stein, 1991). The areas involved in eye movement control are the same as those involved in visuospatial attention. Thus it is not surprising that dyslexics are impaired on a range of spatial

attention tasks including covert orienting; perceptual grouping, visual search, and the inhibition of unattended stimuli (Stein & Walsh, 1997). Patients with acquired dyslexia also tend to show eye movement abnormalities, left neglect, inability to distinguish between rotated letters, crowding effects, and reading problems (see Stein & Walsh, 1997). Stein (1989) proposed that dyslexics have disordered visuospatial function resulting from an abnormality of the RH. The importance of spatial attention to reading and the relationship between spatial attention and dyslexia is discussed in more detail in Chapter 3.

A Multi-modal Sensory Deficit.

The PPC acts as a multimodal sensorimotor association area and receives input from a large number of sources including the somesthetic, proprioceptive, visual, oculomotor, motor, and motivational systems. As previously mentioned, it has been proposed that both the auditory and visual problems in dyslexia can be accounted for by a generalised temporal processing deficit (Farmer & Klein, 1995; Tallal et al., 1993). Stemming from this hypothesis, it has also been argued that the basis of temporal processing deficits is an M-like impairment that extends to other systems such as the vestibular, somatosensory, motor, and auditory systems (Livingstone et al., 1991; Lovegrove, 1996; Stein & Talcott, 1999).

Dyslexics show differences in temporal processing in the auditory modality for tasks involving temporal order judgements as well as a range of other auditory timing and individuation tasks that do not include an 'order' component. These tasks typically involve rapid and/or brief presentation of sequential stimuli and dynamic stimuli that are changing in real time and thought to measure sensitivity to auditory transients (Dougherty, Cynader, Bjornson, Edgell, & Giaschi, 1998; Hari & Kiesila, 1996; Helenius, Uutela, & Hari, 1999c; McAnally & Stein, 1996; Menell, McAnally, & Stein, 1999; Stein & McAnally, 1995; Witton et al., 1998) (but see Hill, Bailey, Griffiths, & Snowling, 1999). Furthermore, abnormalities of M-like cells in the medial geniculate nucleus of the auditory system have been found in dyslexic brains post

mortem, particularly in the LH (Galaburda, Menard, & Rosen, 1994).

The somatosensory system is also composed of large M-like neurones that signal flutter and vibration in the skin. A reduction in the sensitivity to low frequency mechanical vibration and reduced tactile grating orientation discrimination sensitivity have also been observed in dyslexia (Grant, Zangaladze, Thiagarajah, & Sathian, 1999; Stoodley, Talcott, Carter, Witton, & Stein, 2000). Findings from a recent magnetoencephalography study suggest that adults with dyslexia show abnormal response recovery in the somatosensory cortex of the RH (Renvall, Lehtonen, & Hari, 2005). Other research findings indicate that dyslexics perform differently from normal readers on tasks that tap the temporal properties of the motor system including rhythm tapping (Wolff, 2002; Wolff et al., 1984; Wolff et al., 1990) and musical timing skills (Overy, Nicolson, Fawcett, & Clarke, 2003).

The cerebellum also receives M-efferents from the sensory and motor cortices and is important for controlling eye movements and saccades during reading, and possibly the mental articulation required during grapheme phoneme conversion (Stein, 2001a). The largest output of the PPC is to the contralateral cerebellum thus the left temporoparietal areas project to the right cerebellum and developmental lesions to the right and left cerebellum have been shown to result in literacy and visuospatial difficulties respectively (Scott et al., 2001). As previously mentioned, there is some evidence that cerebellar abnormalities in dyslexia are greater in the RH than the LH (Rae et al., 2002; Rae et al., 1998).

Some research findings provide support for an association between reading ability and *general* temporal processing extending across several modalities (Cestnick, 2001; Laasonen, Service, & Virsu, 2002; Laasonen, Tomma-Halme, Lahti-Nuuttila, Service, & Virsu, 2000; Meyler & Breznitz, 2005; Rose, Feldman, Jankowski, & Futterweit, 1999; Van Ingelghem et al., 2001; Witton et al., 1998). For example, significant correlations have been found between measures of visual (coherent motion) and auditory processing (low frequency discrimination) in both normal readers and dyslexics ($r=0.535$), and nonword error time (combined accuracy and

time) was found to correlate significantly with both auditory ($r=0.603$) and visual thresholds ($r=0.406$) (Witton et al., 1998). Similarly, Van Ingelghem et al. (2001) found that 70% of dyslexic children showed both auditory (gap detection) and visual (double flash detection) temporal processing deficits, and both temporal measures were related to word and pseudoword reading. Cestnick (2001) found impaired performance and strong correlations between visual (Ternus task) and auditory (tone repetition test) temporal processing measures for phonological but not surface dyslexics. Meyler and Breznitz (2005) found that high functioning dyslexic adults showed deficits in intra-modal and cross-modal temporal processing tasks for both verbal and nonverbal stimuli. However, it has also been reported that dyslexics with poor auditory temporal processing show enhanced visual sensitivity suggestive of compensatory processing (Heim, Freeman, Eulitz, & Elbert, 2001). Furthermore, some recent studies have shown that performance on temporal processing tasks contribute little unique variance to phonological measures and word reading among dyslexics (Chiappe, Stringer, Siegel, & Stanovich, 2002; Hulslander et al., 2004).

It has also been proposed that phonological and visual problems occur due to abnormalities in the M-like pathway of the auditory and visual systems respectively (Stein, 2001a). For example, sensitivity to low frequency (2Hz) auditory transients has been shown to correlate more strongly with measures of phonological ability (nonword reading and spoonerism decoding) than orthographic ability (irregular word reading) among groups of unselected children whereas visual motion sensitivity correlates more strongly with orthographic skill (Talcott et al., 1999; Talcott et al., 2000b). However, the findings from other studies employing unselected normal adults (Talcott et al., 2002) and dyslexics (Hulslander et al., 2004) have not supported this association.

It is clear that dyslexics and poor readers differ from normal readers on a range of temporal processing tasks across several modalities suggesting a generalised temporal processing deficit. However, a wide range of tasks and definitions of temporal processing have

been used in the literature and the full nature and extent of temporal processing deficits in dyslexia are yet to be established (for a recent review see Klein, 2002). A brief summary of some of the major research findings has been provided but an in-depth analysis of this literature is beyond the scope of the present review.

Summary

This chapter has summarised the major hypotheses regarding the underlying neurobiological basis for dyslexia. Strong research evidence has been found for atypical cerebral lateralisation and LH linguistic dysfunction in dyslexia. Although a LH linguistic deficit is possibly the most parsimonious explanation with the cognitive phonological deficit hypothesis, dyslexia is also associated with a variety of sensory and perceptual problems and while anatomical abnormalities have been observed in both the sensory and linguistic areas of the brain, the causality of this relationship is difficult to determine. Sensory theories often fail to account for the reading difficulties experienced by dyslexics and it has been argued that sensory problems are a correlate rather than a cause of the disorder. The visual problems experienced in dyslexia have been interpreted to reflect an abnormality of either the magnocellular visual pathway and/or areas of the posterior parietal cortex, particularly in the RH. One common feature of sensory and linguistic explanations is that they both implicate areas of the parietal cortex. Whereas the RH is implicated in visuo-spatial problems, the LH is implicated in linguistic processing problems. Both LH and RH parietal areas make up the posterior attention system and are involved in spatial attention. Chapter 3 explores the neuroanatomical relationship between spatial attention and reading ability in detail.

CHAPTER 3 – ATTENTION AND READING ABILITY

Selective Spatial Attention

Attention refers to the mechanism by which we actively process a limited amount of information from the large amount of information available from our senses, stored memories and representations, and other cognitive processes (Posner & Peterson, 1990; Sternberg, 2003). According to Sternberg (2003), the three main functions of conscious attention are signal detection, selective attention, and divided attention. Signal detection refers to the detection of a particular stimulus and may include the processes of vigilance or active search. Selective attention refers to the active selection of particular sensory inputs, while others are ignored, and divided attention is the capacity to allocate attentional resources to more than one task (Broadbent, 1958). The present review is mainly focused on selective attention and in particular selective spatial attention.

The locus of selective attention is a major theoretical issue in the literature (Yantis & Johnston, 1990). Early selection theorists suggest that selective attention occurs after some parallel preliminary analysis but prior to stimulus identification (e.g., Broadbent, 1958; Kahneman, 1973). In this case, selection of relevant stimuli is based on spatial location or pre-attentive features such as colour, orientation, and motion. In contrast, late selection theorists propose that stimulus identification occurs in parallel across the visual field, particularly for patterns that are well learned such as letters or words (e.g., Allport, 1977; Deutsch & Deutsch, 1963). Selection in this case is based on the relevance of stimuli to current information processing demands and filtering occurs after some perceptual and conceptual processing.

The selection of visual input involves both object-based and space-based attentional mechanisms (Duncan, 1984). According to object-based models of attention, sensory input is pre-attentively segmented into objects on the basis of gestalt principles (e.g., continuity, proximity, similarity, movement), followed by a stage in which focal attention is used to process

objects in more detail. In addition, whereas different features of an object are processed in parallel, different objects are processed serially (for a review see Egeth & Yantis, 1997). In contrast to object-based mechanisms, space-based attentional mechanisms are involved in the selection of particular locations, while other locations are ignored or suppressed.

Spatial attention has been likened to a spotlight (Posner, 1980), a filter channel (LaBerge & Brown, 1989), a gradient of processing efficiency (Shulman, Wilson, & Sheehy, 1985), and a zoom lens (Eriksen & St James, 1986). According to the spotlight model, focal attention is characterised by a spotlight of specific size that can be deployed to particular locations within the visual field resulting in greater efficiency of processing at locations that lie within its beam (James, 1950; Posner, 1980; Posner, Snyder, & Davidson, 1980b). However, it has been shown that attention can be allocated over a narrow or wide range of visual space depending on task demands (Jonides, 1981; LaBerge, 1983; LaBerge & Brown, 1986). Thus according to the zoom lens model, focussed attention is of variable size and efficiency of processing is greater when the size of attentional focus is smaller (Eriksen & St James, 1986). According to gradient models of spatial attention, processing efficiency decreases continuously around the current attentional focus corresponding to a gradient of visual attention (Shulman et al., 1985).

Orienting to spatial locations can also involve both automatic or exogenous and voluntary or endogenous modes of attention (Briand & Klein, 1987; Jonides, 1981; Müller & Rabbitt, 1989; Posner, 1980). Endogenous or goal-directed attention refers to a state of attentional readiness that is purposeful on the part of the observer and typically based on task demands. In contrast, exogenous or stimulus-driven attention is summoned automatically by salient features or stimulus onsets (Egeth & Yantis, 1997).

Another important role of selective attention is to filter out input that is currently irrelevant (Pashler, 1998). Input selection or the filtering of irrelevant input can be investigated using interference paradigms such as the Stroop paradigm (Stroop, 1935) or the Eriksen flanker task (Eriksen & Eriksen, 1974). For example, in the flanker task, it takes longer to identify a

central stimulus in the presence of incompatible relative to compatible distractor stimuli. However, this interference can be reduced by top down attentional control suggesting that voluntary attention can mediate the effects of stimulus driven attention (LaBerge, Brown, Carter, Bash, & Hartley, 1991; Yantis & Johnston, 1990).

Other dynamic models of attention have been developed to describe the nature of attentional mechanisms involved in visual search (Duncan & Humphreys, 1989; Treisman & Gelade, 1980; Wolfe, Cave, & Franzel, 1989) and shape identification (LaBerge & Brown, 1989). Visual search models propose that pre-attentive parallel analysis of visual stimuli is followed by a focussed mode of attention in which features are conjoined under the control of spatial attention (Treisman & Gelade, 1980). According to Wolfe's Guided search model attention is directed towards objects as a function of both goal-directed and stimulus directed attentional control (Wolfe, 1994). According to Laberge and Brown (1989), depending on task demands, attention can be allocated over a narrow or diffuse area of visual space. This model is discussed further below in relation to the role of attention in reading.

Attentional Networks in the Brain

Three distinct subsystems of the brain have been proposed to account for the attentional processes of orienting, detecting, and alerting (for reviews see Posner, 2004; Posner & Peterson, 1990). The Alerting System is involved in maintaining an alert or vigilant state and depends upon the norepinephrine pathways that arise in the locus coeruleus and is more strongly lateralised to the RH. The Anterior Attentional Network is involved in executive control and the detection of task relevant stimuli, and is thought to be subserved by frontal areas such as the anterior cingulate gyrus and the lateral prefrontal cortex. The anterior cingulate gyrus is activated during semantic processing, target detection, and the evaluation of multiple attributes (conjunctions) such as colour, form, motion, or word semantics. The anterior cingulate is also thought to monitor conflicting information and acts on the dorsolateral prefrontal cortex in order

to increase selective attention to task relevant and suppress task irrelevant information (Weissman, Gopalakrishnan, Hazlett, & Woldorff, 2005). The Posterior Attentional Network, or orienting system, is activated during tasks in which spatial attention must be disengaged, shifted, or focussed and is subserved by areas of the PPC, the superior colliculus, and pulvinar of the thalamus respectively (Posner & Peterson, 1990).

The PPC is thought to release attention from its current focus and to signal the midbrain (superior colliculus) to move a 'spotlight' of attention to a new location. Patients with lesions to the superior colliculus show disruption in both eye movements and covert shifts of attention, and tend to lose preference for novelty (inhibition of return) (Posner & Cohen, 1984). The pulvinar of the thalamus selects and enhances the contents of the attended area to aid target detection and response generation in anterior areas (Corbetta & Shulman, 2002). Patients with lesions of the thalamus have difficulty separating targets from surrounding distractors, particularly in the contralesional visual field. In addition, monkeys with lesions to the pulvinar are faster to respond to targets appearing on the side of the lesion when previously cued to the side opposite the lesion, suggesting that the contralateral cue is not effective in engaging attention (Posner & Peterson, 1990).

Although orienting, alerting, and executive control are mediated by relatively independent systems (e.g., Fan et al., 2002), there is also considerable interaction between them (Callejas et al., 2004). The alerting system may act on the Posterior Attention Network to accelerate the process of visual orienting (Callejas et al., 2004) and may also act on the Anterior Attention Network to decrease cognitive activity in order to aid signal detection (Posner, 1994; Callejas et al., 2004). There is also some evidence for an interaction between orienting and executive function. For example, when attention is oriented away from target locations the spatial Stroop effect (Funez & Lupianez, 2003) and the flanker effect are larger (Callejas et al., 2004).

Another neuroanatomical model of attention proposes two distinct neural networks for the control of visual attention (see Corbetta & Shulman, 2002). The selection of sensory information and responses is thought to be subserved by the PPC along the Intraparietal Sulcus (IPs) and the frontal cortex near the Frontal Eye Fields (FEF), whereas the detection of task relevant sensory events is mediated by RH areas including the temporo-parietal junction (TPJ) and the ventral frontal cortex (VFC). The former IPS-FEF network is effectively similar to the combined anterior and posterior networks described by Posner and Peterson (1990). The latter TPJ-VFC system is similar to the alerting network described by Posner and is involved in orienting attention to task relevant and low probability expected or unexpected events. This system acts as a circuit breaker for the IPs-FEF network in order to interrupt ongoing cognitive activity. The IPs is thought to indicate the behavioural relevance of stimuli to the TPJ, either directly or through top-down modulation of the visual cortex and the frontal aspect of the ventral network may be specifically related to the processing of novel stimuli. Visual attention is modulated by both bottom-up factors such as sensory stimulation and top-down factors such as goals, expectation, and knowledge (attentional sets). The IPs-FEF pathway is involved in the generation of attentional sets and goal-directed response selection (Corbetta & Shulman, 2002). Salience maps in this system sum the contribution of top-down and bottom-up factors of visual objects and determine which are selected for recognition and action.

The system involved in covert orienting of spatial attention is dominated by the RH and is similar to the network involved in normal eye movement control (Nobre, 2001; Nobre, Gitelman, Dias, & Mesulam, 2000). However, attention to object or feature information as opposed to spatial information is associated with increased parietal-frontal activation in the LH (Nobre, 2001). A similar increase in LH activation has also been observed during motor attention and in temporal rather than spatial orienting. Corbetta et al. (2000) found that IPs is activated when attention is voluntarily maintained at cued locations, however, when attention is drawn to unattended locations by cues, the TPJ is preferentially activated. However, Nobre et al.

(2001) argue that the findings of imaging studies generally support the view that the parietal-frontal system is involved in both exogenous and endogenous modes of attention. Though there is some evidence for greater involvement of LH parietal areas in voluntary orienting, exogenous attention is thought to be more strongly lateralised to the RH (Nobre, 2001).

The Real Neural Architecture (RNA) model outlines the possible mechanisms by which subcortical structures (the pulvinar and the superior colliculus) interact with the cortical circuits involved in attention to produce a spotlighting function (Shipp, 2004). The two major cortical connections to the pulvinar are the ‘dorsal’ parietal-supero-temporal and the ‘ventral’ occipito-infero-temporal pathways. According to this model, topographic maps within the ventral visual processing stream are fused to form global and secondary visual maps in the pulvinar and top-down signals from the fronto-parietal network are relayed onto this map through the superior colliculus. This circuitry creates a spotlight of attention that modulates the excitability of visual maps within the ventral visual processing pathway. According to this model, exogenous attention, which is based on saliency, is determined by colliculo-thalamic interactions along the ventral visual pathway without interaction with the fronto-parietal attention system. However, top down inputs from higher order frontal and infero-temporal areas can modulate the saliency attached to particular features and the fronto-parietal pathway can use these saliency signals to exert control over the attentional focus, possibly through the pathway from the superior colliculus to the thalamus. Thus, according to this model, the pulvinar plays a vital role in combining both bottom-up and top-down influences to compute the salience of features in the visual field. This theory is supported by findings that incoming visual information is modulated by attention as early as the primary visual cortex in both Macaque monkeys (Vidyasagar, 1998) and humans (e.g., Martinez et al., 1999; Martinez et al., 2001).

Spatial Attention and Reading Impairment

The findings of a large number of studies indicate that children with dyslexia show a selective spatial attention deficit. Dyslexics differ from controls on tasks that measure attentional processes such as visual search, attentional dwell and capture, covert orienting, inhibition of distracting stimuli, the spatial scale of attention, and hemispheric control over attention. Together these findings have often been interpreted to indicate a RH parietal deficit. However, there is little consensus on the causal relationship between attentional difficulties and reading disability and little empirical evidence for the specific neuroanatomical mechanisms that are involved. The following section reviews this area of research in detail.

Visual Search

According to feature integration theory, visual search for a single feature involves a single parallel process such that the attended feature ‘pops out’ and search times are independent of the number of distractor items in a set. In contrast, visual search for conjunctions of features requires the binding of features from feature maps at one location at a time, under the control of serial focal attention (Treisman & Gelade, 1980). According to more recent modifications of feature integration theory, focal attention is guided by pre-attentive segmentation of the visual field and both excitatory (Wolfe et al., 1989) and inhibitory (Treisman & Sato, 1990) guidance are used to control search for feature conjunctions.

One possible role for spatial attention during reading is the conjoining or integration of features of letters and words into meaningful objects. Poor readers are slower than normal readers on visual search tasks requiring serial letter scanning and cancellation tasks (Casco, Tressoldi, & Dellantonio, 1998; Williams, Brannan, & Lartigue, 1987; Williams, May, Solman, & Zhou, 1995). Other studies have investigated the relationship between reading ability and visual search using traditional visual search paradigms that are less reading-like in nature (Buchholz & McKone, 2004; Casco & Prunetti, 1996; Hayduk et al., 1996; Illes, Walsh, &

Richards, 2000; Ruddock, 1991; Vidyasagar & Pammer, 1999). Some researchers have found differences between dyslexics and controls on tasks requiring simple feature searches (e.g., Ruddock, 1991), while other research suggests that dyslexics differ in their performance on conjunction search tasks but not tasks involving the search for single features (Buchholz & McKone, 2004; Casco & Prunetti, 1996; Illes et al., 2000). Further, some studies have demonstrated a greater decrease in performance as a function of distractor set-size for conjunction tasks among both dyslexic children (Vidyasagar & Pammer, 1999) and adults with poor phonological processing skills (Buchholz & McKone, 2004).

Together the above findings have been interpreted to indicate differences in the functioning of an attentional spotlight mechanism that is mediated by the M pathway (e.g., Vidyasagar & Pammer, 1999). Consistent with this proposal, dyslexics with elevated coherent motion thresholds differ more from controls on conjunction search tasks than those without a concurrent motion coherence deficit (Illes et al., 2000). However, Buchholz and McKone (2004) found that performance on a visual search task and not the M-mediated ‘frequency doubling illusion’ was related to measures of phonological awareness in adult dyslexics with persistent phonological processing deficits.

The attentional mechanisms involved in visual search may be similar to those involved in reading. For example, the processes that guide excitation of target locations and inhibition of distractor locations may be similar to those that facilitate the identification of attended words or letters and the inhibition of unattended letters or words during reading. Furthermore, both require the rapid integration of information across space (Illes et al., 2000) and the conjoining of stimulus features into perceptual objects. Lesions and transcranial magnetic stimulation to the PPC have been shown to produce impairment in conjunction searches consistent with this area’s involvement in the allocation of spatial attention (e.g., Arguin, Joanette, & Cavanaugh, 1993; Ashbridge, Walsh, & Cowey, 1997; Corbetta, Shulman, Miezin, & Petersen, 1995; Friedman-Hill, Robertson, & Treisman, 1995).

Attentional Dwell and Capture

When subjects are asked to identify the second of two rapidly presented stimuli a decrease in accuracy occurs as a function of decreasing Stimulus Onset Asynchrony (SOA) between the stimuli. This attentional blink phenomenon is thought to index the time required to reallocate attentional resources. Dyslexics show a prolonged attentional blink relative to controls (e.g., Hari, Valta, & Uutela, 1999; Visser, Boden, & Giaschi, 2004). For example, Hari et al. (1999) found that dyslexics showed a significantly longer attentional blink to letter stimuli as a function of SOA, suggesting a prolonged attentional dwell time, due to difficulty in disengaging or re-engaging attention to the first and second stimulus respectively. Visser, Boden, and Giaschi (2004) found that when targets were presented at the same spatial location, dyslexics showed a greater attentional blink than chronological age but not reading age matched controls, however, when targets were presented at different spatial locations (requiring both temporal and spatial reallocation of attention) performance of dyslexics was marginally worse than reading age matched controls, suggesting a deficit in rapidly allocating attention over time and space (Visser et al., 2004). Another experiment employing visual temporal order judgment and ‘line motion illusion’ tasks found that adult dyslexics showed sluggish attentional capture in both visual fields, coupled with evidence for a RVF attentional bias and LVF mini-neglect (Hari, Renvall, & Tanskanen, 2001).

A prolonged attentional blink in dyslexia may be related to RH dysfunction, as lesion and MRI research indicates that RH parietal areas are involved in the phenomena (Hari et al., 2001). Furthermore, areas of the PPC are thought to control the process of disengaging attention from areas of the visual field (Posner & Peterson, 1990). Hari and Renvall (2001) proposed a ‘sluggish attentional shift’ theory of dyslexia. According to this theory, a RH parietal deficit results in slow attentional capture and prolonged attentional dwell time which accounts for

impairment of processing rapid stimulus sequences in all sensory modalities and the phonological processing deficits observed in dyslexia (Hari & Renvall, 2001).

Steinman, Steinman and Lehmkuhle (1997) found that the M system plays an important role in the automatic allocation of attention in the line motion task. For example, cues that preferentially stimulate the M pathway have a stronger and more rapid attentional response than those that preferentially stimulate the P pathway. Following on from this, Steinman measured spatiotemporal attentional response functions using the line-motion task in compensated adult dyslexics and controls (Steinman, Steinman, & Garzia, 1998). Their findings indicated that dyslexics showed a narrower attentional focus and a greater area of inhibition outside the area of attentional focus, particularly to the right of fixation. It was argued that a deficit in M-mediated spatial attention affects the planning of saccadic eye movements during reading. However, this interpretation does not explain why dyslexics have difficulty reading single words that are not embedded within text and it has also been argued that eye movement deficits are a consequence rather than a cause of reading disability (Morris & Rayner, 1991).

Covert Orienting

Covert orienting or the shifting of visual attention without overt eye or head movements can be investigated using valid and invalid spatial cues that correctly or incorrectly predict the location of a subsequent target (Posner, 1980; Posner et al., 1980b). Compared to neutral spatial cues, valid and invalid cues result in behavioural (RT and accuracy) benefits and costs respectively. RT benefits are thought to result from perceptual facilitation due to both faster intake of information and better perceptual representations or signal to noise ratio (see Carrasco & McElree, 2001; Mangun, 1995). Orienting to spatial locations may involve both automatic or exogenous and voluntary or endogenous modes of attention (Müller & Rabbitt, 1989). Central or symbolic cues are thought to generate a voluntary shift in attention whereas peripheral cues are thought to engage an exogenous mechanism that produces facilitation of RT with SOAs as early

as 50-100ms. However, depending on task demands such as SOA and the percentage of valid trials (cue informativeness), inhibition of return (IOR) may occur such that participants are slower to respond to targets at cued than uncued locations (Klein, 2000; Müller & Rabbitt, 1989; Posner & Cohen, 1984).

A large number of studies have investigated covert orienting of attention among dyslexics or poor readers (Brannan & Williams, 1987; Facoetti et al., 2003b; Facoetti, Lorusso, Paganoni, Umiltà, & Mascetti, 2003c; Facoetti, Paganoni, Turatto, Marzola, & Mascetti, 2000b; Facoetti, Turatto, Lorusso, & Mascetti, 2001; Harter, Anillo-Vento, & Wood, 1989; Heiervang & Hugdahl, 2003; Jonkman, Licht, Bakker, & Van den Broek-Sandmann, 1992; Roach & Hogben, 2004). The findings of some studies indicate greater differences between dyslexics and controls at short rather than long SOAs and for peripheral rather than central cues, suggestive of an automatic orienting deficit in dyslexia (Brannan & Williams, 1987; Facoetti et al., 2000b; Heiervang & Hugdahl, 2003). The relationship between covert orienting and reading ability has also been demonstrated in unselected readers. For example, Kinsey, Rose, Hansen, Richardson, and Stein (2004) found that a brief spatial cue was more effective in drawing attention away from or towards a visual target in a cued motion coherence task among higher ranked in comparison to lower ranked readers. Furthermore, performance on valid trials contributed significant but small amounts of unique variance to the prediction of both nonword (11%) and irregular word reading (8%). Similarly the difference between valid and invalid trials contributed unique variance to the prediction of non-word reading accuracy (12%).

Differences in inhibitory processing have also been observed between dyslexics and controls during the performance of covert orienting tasks. For example, using uninformative peripheral cues, Facoetti et al. (2003b) found that dyslexics showed a lack of facilitation at short SOAs coupled with a lack of inhibition of return at longer SOAs. In a similar experiment that included neutral as well as valid and invalid trials, Facoetti et al. (2003c) found that whereas normal readers showed both facilitation (benefits) and inhibition (costs) relative to neutral trials,

dyslexic children showed benefits but not costs, suggesting a deficit in the suppression or inhibition of unattended stimuli. It was also shown that visual hemisphere specific stimulation but not speech training produced a significant decrease in reading speed and accuracy and an increase in the inhibition effect for dyslexics such that an increase in RT was observed for invalid trials post training.

The findings of other covert orienting studies indicate visual field differences between dyslexics and controls. For example, Brannan and Williams (1987) found that good readers and adults but not dyslexics showed an accuracy advantage for RVF in comparison to LVF trials in a covert orienting task involving letter stimuli. Under peripheral cueing conditions, Facoetti et al. (2001) found that normal readers showed an overall effect of cue regardless of visual field but dyslexics show an effect of cue for LVF but not RVF trials. A similar pattern of results was found for the central cue condition, though the difference between the visual fields was reduced. This finding was interpreted to reflect a lack of inhibition of the unattended contralateral visual field, such that suppression of the RVF was absent when the cue was presented in the LVF (Facoetti et al., 2001). In a further investigation it was found that right attentional inhibition characterised by decreased RT for RVF invalid trials was present in dyslexic children with impaired nonword reading, but not in dyslexics without impaired nonword reading or controls (Facoetti et al., 2006). Further, dyslexics with a nonword reading deficit also showed longer reaction times to invalid trials in the LVF compared to the other two groups.

The differences observed between dyslexics and controls on covert orienting tasks have often been interpreted to reflect differences in the functioning of the M-mediated PPC, particularly in the RH (e.g., Facoetti et al., 2000b; Facoetti et al., 2001). This is consistent with research showing that damage to the parietal cortex results in less inhibition of the opposite hemisphere (Ro, Cohen, Ivry, & Rafal, 1998). Further, visual attention is predominantly captured by cues that preferentially stimulate the M pathway (Steinman et al., 1997).

Consistent with the multimodal version of the M-hypothesis and the temporal processing hypothesis, there is also evidence that the covert orienting deficits observed among dyslexics extend to the auditory modality (Facoetti, Lorusso, Cattaneo, Galli, & Massimo, 2005; Facoetti et al., 2003b). For example, dyslexics fail to show facilitation of auditory spatial attention at short SOA (100ms) and inhibition of return at longer SOAs (250ms) (Facoetti et al., 2003b). In a similar study, chronological age and reading age matched control groups, showed a reduction in visual and auditory cueing at long SOAs, whereas the opposite pattern was observed for dyslexics (Facoetti et al., 2005). These findings are consistent with the proposal that dyslexics suffer from a multimodal attentional deficit (Hari & Renvall, 2001; Vidyasagar, 1999).

Several lines of research indicate that areas of the PPC are involved in spatial orienting in both the visual and auditory modality. For example, parietal lesions produce impairment on both auditory and visual covert orienting tasks for contralesional targets preceded by an uninformative invalid cue, indicating impairment in the mechanism that disengages attention from the ipsilesional to the contralesional side of space (e.g., Farah, Wong, Monheit, & Morrow, 1989). In a recent functional MRI study, a right lateralised multimodal network was found to respond to sensory changes in the visual, auditory, and tactile modalities (Downar, Crawley, Mikulis, & Davis, 2000). This network included the temporo-parietal junction, the middle temporal gyrus, the insula, the right inferior frontal gyrus, and the left anterior cingulate and supplementary motor areas.

The Spatial Scale of Attention

While covert orienting studies have investigated how attention moves in the visual field, other studies have been more concerned with the ability of the visual system to vary the spatial extent of attentional focus. Focussing is thought to consist of two stages, an automatic process triggered by the stimulus onset that adjusts the size of attentional focus and a voluntary process that maintains the size of attentional focus (Benso, Turatto, Mascetti, & Umiltà, 1998; Turatto et

al., 2000). Top-down attentional control can be used to actively focus on an object, but the bottom-up 'focusing reflex' is activated by stimulus onsets and can not necessarily be avoided even when another object is the voluntary focus of attention. Consistent with the zoom lens model of attention, the findings of research employing cue-size paradigms indicate an inverse relationship between the size of attentional focus and processing efficiency (Castiello & Umiltà, 1990; Castiello & Umiltà, 1992; Eriksen & St James, 1986; Henderson, 1991).

However, researchers who have investigated attentional focussing in dyslexia have revealed conflicting findings. Facoetti et al. (2000b) found that dyslexics showed a cue size effect at short (99ms) but not long (504ms) SOAs, suggesting that they were unable to maintain active focussing over time and had shifted to a more distributed and less efficient visual processing mode. However, Facoetti et al. (2003a) found that normally reading children showed a cue size effect at both SOAs, whereas dyslexic children showed a cue size effect at the long (500ms) but not the short (100ms) SOA providing further evidence of an automatic orienting deficit in dyslexia. The authors suggested that the different findings of these two studies could be due to task difficulty, perceptual load, or the allocation of processing resources (Facoetti et al., 2003a).

Other research findings indicate that poor readers are inclined towards a more diffuse allocation of attention or an inclination for global processing (Facoetti & Molteni, 2001; Facoetti, Paganoni, & Lorusso, 2000a; von Karolyi, 2001; von Karolyi, Winner, Gray, & Sherman, 2003; Williams & Bologna, 1985). Facoetti, Paganoni, and Lorusso (2000a) investigated the spatial distribution or gradient of visual attention in dyslexic children using a dot detection task in which targets were presented at eccentricities that fell within or outside a circular focussing cue. Normal children showed an increase in RT with increasing eccentricity, whereas dyslexics showed a more diffuse distribution of visual processing resources. A second experiment used a visual search task in which targets defined by a single feature were presented in a circular array with a variable number of distractors. Dyslexics showed longer RT in

comparison to controls, but a smaller increase in RT with increasing number of distractors relative to normal readers, suggestive of diffuse parallel processing. Together, these data were explained in terms of difficulty in narrowing the focus of attention and a natural spontaneous tendency to process visual features using a distributed mode of visual attention. It was argued that this may affect the facilitation of relevant information and the suppression or inhibition of irrelevant information in the visual field during reading (Facoetti et al., 2000a). Dyslexics are also better than controls at recognising impossible figures in a global visual-spatial task suggesting enhanced ability to process information in a global or holistic fashion (von Karolyi, 2001; von Karolyi et al., 2003). Further, Williams and Bologna (1985) found that poor readers showed greater perceptual grouping effects in a selective attention task (speeded card sorting) than good readers and a significant negative correlation between perceptual grouping and reading scores ($r=-0.48$). They suggested that poor readers are less able to selectively attend to elements within the same perceptual unit indicating an inclination for global or holistic rather than analytic or local processing. Similarly, Stephen and Williams (1986, cited in Brannan & Williams, 1987) used an object superiority paradigm and found that normal readers accuracy was linked to the perceived depth of surrounding context pattern whereas poor readers based their judgements on perceived connectedness of surrounding context.

Reading requires the allocation of spatial attention to both individual letters and whole words comprised of individual elements. As such, the allocation of attention during reading may be similar to processing hierarchical stimuli in global/local processing paradigms (Kimchi, 1992; Navon, 1977). However, few studies have investigated visual processing in dyslexia using traditional global/local paradigms. Williams and LeCluyse (1990) cite an in press publication of Williams and Brannan in which there was no effect of consistency for local letter identification among disabled readers. However, when the stimulus was blurred, the RT of disabled readers became faster and the consistency effect appeared. This was argued to occur due to the re-established temporal relationship between the transient (M) and sustained (P) visual processing

systems. Keen and Lovegrove (2000) investigated the effects of size and retinal eccentricity on global/local processing in dyslexic children and chronological age and reading age matched controls. Considering that dyslexia is associated with a deficit in the M visual processing stream it was argued that this would transfer to a deficit in global relative to local processing. However, whereas dyslexics showed longer RT in comparison to chronological age matched controls and shorter RT in comparison to reading age matched controls, there were no differences in global precedence or eccentricity effects. It was argued that dyslexics are slow at processing visual information which affects the rapid processing of peripheral information and its integration with information from fixation during reading (Keen & Lovegrove, 2000).

Together the findings of diffuse attentional processing in dyslexia are consistent with research showing that dyslexics may use larger orthographic units when reading words (e.g., van der Leij & van Daal, 1999) and may compensate for phonological decoding deficits by employing a visual or orthographic strategy (e.g., Marcel et al., 1974; Waldie, 2002). However, it should be noted that the RH is preferentially involved in the global or holistic processing of visual stimuli (Martinez et al., 1997), and that global processing is associated with low spatial frequencies suggesting involvement of the M pathway (Badcock, Whitworth, & Badcock, 1990; Shulman & Wilson, 1987b). Thus the suggestion that dyslexics have a propensity for global processing is not necessarily consistent with the RH deficit hypothesis or the magnocellular hypothesis.

Inhibition of Unattended Stimuli

Some research findings indicate that dyslexics have difficulty inhibiting distracting visual information. As previously mentioned both dyslexic children (Vidyasagar & Pammer, 1999) and adults with poor phonological processing skills (Buchholz & McKone, 2004) show a greater increase in visual search times as a function of distractor set size which could be due to a lack of inhibition of irrelevant stimuli. Gernsbacher (1993) found that poor adult readers are less

able to suppress irrelevant information including incorrect forms of phonemes, inappropriate meanings of ambiguous words, typical but absent objects in scenes, and words superimposed on pictures. Although it is possible that these effects occurred due to less *enhancement* of contextually appropriate information, Gernsbacher (1993) reports evidence to suggest that it is the general ability to suppress inappropriate information that caused these differences between good and poor readers. Dyslexic children do not differ from controls in the identification of single letters in foveal and parafoveal vision, but show poorer performance for both word recognition and the identification of letters embedded in other letters (e.g., the identification of the letter 'a' in 'xax') (Bouma & Legein, 1977). Similarly, Brosnan et al. (2002) found that both compensated dyslexic adults and dyslexic children showed lower performance relative to controls on a group-embedded figures test requiring the inhibition of distracting stimuli. These findings were thought to indicate an executive function deficit involving the left prefrontal cortex.

Other research findings indicate that dyslexics differ from controls on measures of inhibition such as the Stroop task (Everatt et al., 1999b) and flanker task. Klein and D'Entremont (1999) used a flanker filtering task in which participants had to identify one of two digits which were flanked by incompatible or compatible flanking distractors at different eccentricities. Adult psychology students who were poor readers did not show the same decrease in the size of the flanker effect as a function of flanker eccentricity as good readers suggesting less interference at short distances and more interference at far distances. Facoetti and Turatto (2000) investigated interference in dyslexia using a flanker task in which response compatible or incompatible distractors were presented adjacent to a target stimulus. Whereas normal readers exhibited a symmetrical flanker effect, dyslexics exhibited a reduced flanker effect in the LVF and a strong flanker effect in the RVF (Facoetti & Turatto, 2000). It was concluded that visual information presented in RVF was not filtered efficiently resulting in distraction by letters or words presented to the right of fixation.

Bednarek et al. (2004) investigated the processes of alerting, orienting, and inhibition (or resolution of conflict) among Spanish dyslexics and normally reading children using a cueing task in which targets were surrounded by compatible and incompatible flanking stimuli. Dyslexics were found to be impaired relative to controls on accuracy and RT measures when targets were flanked by incompatible flankers suggesting a deficit in executive control rather than orienting or alerting. This flanker effect was found to correlate significantly with measures of reading and writing ($r=.34-.47$). It was argued that the neuro-physiological origin of this effect could be either executive function mediated by the prefrontal cortex, or attentional processes mediated by the PPC, and including the magnocellular visual processing stream which has been linked to the identification of flanker stimuli.

However, Roach and Hogben (2004) investigated spatial cueing with a single fixation search task in five adult dyslexics selected on the basis of nonword reading ability. The task measured thresholds for orientation discrimination in which subjects had to determine the location of a Gabor patch relative to a variable number of distractor patches presented concentrically around a central fixation cross. Cueing the location of a target removed much of the increase in RT associated with increasing numbers of distractors for normal readers but not for dyslexics. However, there were no differences between the groups on tasks that tap M functioning (flicker contrast sensitivity and global dot motion) suggesting that the spatial cueing deficit is not necessarily the product of a magnocellular deficit (Roach & Hogben, 2004).

The findings of a series of experiments investigating peripheral vision and lateral masking effects in dyslexia also suggest differences in the suppression of information in the periphery (e.g., Geiger & Lettvin, 1987, 2000; Geiger, Lettvin, & Fahle, 1994; Geiger, Lettvin, & Zagarra-Moran, 1992). The form resolving field is the plot of the probability of recognition of a peripheral letter as a function of its eccentricity to a central letter. Normal readers typically show a monotonic decrease in the recognition of the peripheral letter as a function of eccentricity. Adults (Geiger & Lettvin, 1987; Geiger et al., 1992) and children (Geiger et al.,

1994) with dyslexia show a wider distribution of the form resolving field such that recognition performance is greater at peripheral eccentricities, particularly in the RVF. Several other researchers have replicated these findings (e.g., Dautrich, 1993; Lorusso et al., 2004; Lorusso, Facoetti, Toraldo, & Molteni, 2005; Perry, Dember, Warm, & Sacks, 1989) (but see Bjaalid, Høien, & Lundberg, 1993; Goolkasian & Garver, 1995; Klein, Berry, Briand, D'Entremont, & Farmer, 1990; Slaghuis et al., 1992) and this difference was common to all dyslexic subtypes classified according to Bakker's typology (Lorusso et al., 2004).

According to Geiger and Lettvin (1999), dyslexics experience greater lateral masking for letters presented to the right of fixation and less masking further in the periphery which hinders word identification causing visual confusion. However, it has also been suggested that the form resolving field reflects the spatial distribution of attention and therefore dyslexics have difficulty focussing attention in the centre and inhibiting peripheral visual information (Lorusso et al., 2004). Abnormal interactions between peripheral and foveal vision are consistent with findings that dyslexics make more errors for reading whole lines of text rather than single words (Hill & Lovegrove, 1993; Pepper & Lovegrove, 1999). A case of developmental dyslexia has been reported in which parafoveal stimuli were found to interfere with processing of words in fixation (Rayner, Murphy, Henderson, & Pollatsek, 1989). Some researchers suggest that the abnormal lateral masking function can be explained in terms of interactions between the transient (M) and sustained (P) visual pathways (Slaghuis et al., 1992; Williams et al., 1990). Furthermore, the 'shift effect', in which detectability of foveal targets is reduced by movement in the periphery is thought to be mediated by the M layers of the lateral geniculate nucleus (Cornelissen et al., 1995).

The widening of the form resolving field in dyslexia is observed in the RVF for native English speaking adults and the LVF for native Hebrew speaking adults suggesting that it is related to the direction of reading and represents a learned attentional strategy (Geiger et al., 1992). Geiger and colleagues developed a training regimen for dyslexics that resulted in a

narrowing of the form resolving field in the RVF for dyslexics as well as an improvement in reading (Geiger & Lettvin, 2000; Geiger et al., 1994; Geiger et al., 1992). The training regime involved practicing novel hand-eye coordination tasks (e.g., drawing, painting etc.) and reading through a rectangular window in the right periphery at an eccentricity while looking at a fixation point. Visual hemisphere specific stimulation has also been shown to result in a change in the distribution of the form resolving field in dyslexics providing further support for its learned nature (Lorusso et al., 2005).

Asymmetric Distribution of Spatial Attention in Dyslexia?

Some research findings indicate that dyslexia is associated with an asymmetrical distribution of attention characterised by LVF inattention and RVF distractibility (Eden, Wood, & Stein, 2003; Facoetti & Molteni, 2001; Facoetti & Turatto, 2000; Facoetti et al., 2001; Geiger & Lettvin, 1987; Hari et al., 2001). As previously mentioned, in covert orienting tasks dyslexics (particularly those with a nonword reading deficit) showed a peripheral cuing effect for LVF but not RVF trials, suggesting a lack of inhibition of the unattended RVF when attention is cued to the LVF (Facoetti et al., 2001; Facoetti et al., 2006). Dyslexics also show a broadening of attention in the periphery of the RVF as measured by the form resolving field (Geiger & Lettvin, 1987, 2000; Lorusso et al., 2004), and a RVF attentional bias during visual temporal order judgement and line motion illusion tasks, particularly at short delays (Hari et al., 2001). Facoetti and Turatto (2000) found that whereas normal readers exhibited a symmetrical flanker effect, dyslexics exhibited a reduced flanker effect in the LVF and a strong flanker effect in the RVF (Facoetti & Turatto, 2000). It was suggested that dyslexics may be distracted by letters or words presented to the right of fixation and that inattention to the LVF might play a role in regressive saccades.

In addition to these findings, Facoetti and Molteni (2001) found that dyslexic children showed slower reaction times in comparison to controls but a normal target eccentricity effect

for LVF trials, but not RVF trials. Facoetti et al. (2006) found that dyslexics showed increased RT to invalid trials in the LVF during covert orienting tasks. Eden, Wood, and Stein (2003) found evidence for LVF neglect among dyslexic children using a clock drawing task. Furthermore, children with reading problems and unstable vergence control make more errors locating targets in the LVF than RVF (Stein, Riddell, & Fowler, 1989), and smooth pursuit eye movements are particularly poor when moving in a left-to-right direction (Eden et al., 1994).

Together the findings of asymmetrical attention in dyslexia are considered to be consistent with RH parietal dysfunction (Facoetti & Turatto, 2000; Facoetti et al., 2001; Hari & Renvall, 2001; Hari et al., 2001). Unilateral neglect of contralesional space following posterior parietal damage is a well known neuropsychological phenomenon that is more often associated with RH than LH damage (Posner, Walker, Friedrich, & Rafal, 1984). Some patients are selectively impaired at attending to a particular spatial location, whereas others may be selectively impaired at disengaging attention from a particular object (Riddoch, Humphreys, Cleton, & Fery, 1990). A slowing of temporal order judgements has also been observed in neglect patients (see Hari et al., 2001), and patients with temporoparietal lesions show a reduction in flanker effect in contralesional visual field, and a larger effect in the ipsilesional visual field (Ro et al., 1998).

Kinsbourne (1970) argued that unilateral lesions disrupt the mutual inhibitory interaction between the hemispheres, biasing attention to one side. Further, RH areas of the PPC are proposed to contain bilateral receptive fields that represent both visual hemi-fields, whereas the LH has contralateral receptive fields that represent only the RVF (see Hillis et al., 2005). The areas most commonly implicated in neglect are areas of the PPC including the inferior parietal lobule and the temporo-parietal-junction. It has been argued that right temporo-parietal lesions cause inactivation of the right parietal-frontal, and in accordance with hemispheric models of visual orienting (e.g., Kinsbourne, 1970), this inactivation may also result in relative hyper-activation of the left parietal-frontal network (see Corbetta & Shulman, 2002). For example,

patients with RH parietal lesions show increased detection times in the LVF and/or decreased detection speeds in the RVF (Posner et al., 1984; Posner, Walker, Friedrich, & Rafal, 1987; Smania et al., 1998).

Parietal damage also causes several types of neglect dyslexia (Haywood, 2001; Haywood & Coltheart, 2000; Riddoch et al., 1990). Patients may show neglect at the retinocentric feature level, the stimulus centred letter level, and the word centred graphemic level (Haywood & Coltheart, 2000). Hillis et al. (2005) found that left viewer centred neglect dyslexia was associated with dysfunction of the right inferior parietal lobule. In contrast, damage to right temporal regions caused stimulus centred neglect. These findings are consistent with the notion that both of these RH areas attend to both sides of space, whereas the LH predominantly attends to the right side of space. It was also suggested that the dorsal visual processing stream is involved in the viewer centred representations that guide goal directed movements such as the extra-ocular movements involved in reading, whereas the ventral visual processing stream is involved in the stimulus centred and object centred representations involved in word recognition.

As previously mentioned, the PPC receives predominantly magnocellular input, and the M system projects preferentially to the RH. There is also evidence that the M system is involved in the automatic capture of attention (Steinman et al., 1997) and the allocation of attention during the identification of letters in flanker tasks (Omtzigt & Hendriks, 2004; Omtzigt, Hendriks, & Kolk, 2002). Thus it possible that damage to the M system could cause deficits in spatial attention.

A causal link between spatial attention and dyslexia?

Facoetti et al. (2005) found that dyslexic children differed from both reading age and chronological age matched controls in covert orienting tasks suggesting a causal role of spatial attention in the development of reading disability. The findings of some rehabilitation studies also provide support for the causal nature of spatial attention (Facoetti et al., 2003c; Geiger &

Lettvin, 1999; Lorusso et al., 2005; Solan, Shelley-Tremblay, Ficarra, Silverman, & Larson, 2002). For example, Facoetti et al. (2003c) found that visual hemisphere specific stimulation, but not speech training, produced a significant improvement in reading speed and accuracy and a reversal in the attentional inhibition effect observed for dyslexics such that an increase in RT was observed for invalid trials post training. Similarly, visual hemisphere specific stimulation has been shown to result in a change in the distribution of the form resolving field in dyslexics (Lorusso et al., 2005). In accordance with Kinsbourne's theory of attentional control (Kinsbourne, 1970), hemisphere specific stimulation increases activation in the contralateral hemisphere and decreases activation in the ipsilateral hemisphere leading to a redistribution of processing resources (Lorusso et al., 2006). Solan et al. (2002) investigated the effect of computer based visual attention therapy on reading comprehension in children with moderate reading disability. The therapy aimed to stimulate sustained and selective (shifting and focussing) attention and included perceptual accuracy, visual efficiency, visual search, visual scan, and visual span programs. Compared to a no-treatment control group, those who received attention therapy had improved on measures of attention (attention scales of cognitive assessment system) and reading comprehension (see also Thomson et al., 2005 for similar findings).

However, it is possible that spatial attention difficulties are a correlate rather than a cause of reading disability and it is also possible that spatial attention represents a secondary compensatory mechanism that determines the extent of reading difficulties above a core phonological deficit. Furthermore, children with ADHD also have problems with limiting spatial attention to particular locations and selectively processing and inhibiting relevant and irrelevant information as measured by 'flanker' and visual search tasks (Shalev & Tsal, 2003). Thus it is possible that the attentional difficulties experienced by dyslexics are due to co-morbid ADHD symptoms.

The role of spatial attention in word recognition and reading disability

A potential problem for attentional explanations of dyslexia is their ability to explain the heterogeneity observed among dyslexic populations and to describe the precise mechanisms by which spatial attention deficits produce the reading impairments observed. Neither of the major reading theories consider spatial attention to be an integral part of the reading process (Coltheart et al., 1993; Coltheart et al., 2001; Harm & Seidenberg, 1999; Plaut et al., 1996; Seidenberg & McClelland, 1989). However, other theories of word recognition (Ans, Carbonnel, & Valdois, 1998; Behrmann, Moscovitch, & Mozer, 1991; LaBerge & Brown, 1989; LaBerge & Samuels, 1974; Whitney, 2001; Whitney & Lavidor, 2004), and eye movement control (Inhoff, Pollatsek, Posner, & Rayner, 1989; Morrison, 1984; Pollatsek, Rayner, Fischer, & Reichle, 1999; Reichle, Pollatsek, Fisher, & Rayner, 1998) emphasise the role of attention in the reading process. The spatial orienting system plays an important part in the generation and control of saccades and fixations during reading and may act as a filtering mechanism to inhibit letters and words that are not currently being fixated (Inhoff et al., 1989; Pollatsek et al., 1999). However, the present review will focus on the role of spatial attention in single word identification processes.

Early selection theorists proposed that spatial attention must be focussed on a word prior to recognition. For example, According to feature integration theory, focal attention acts to conjoin elementary features of objects such as words prior to accessing lexical representations (Treisman, 1988; Treisman & Gelade, 1980). Another model proposes that spatial attention controls which spatial locations are allowed to pass from feature to letter level processing and thus assumes that unattended words are not identified (Yantis & Johnston, 1990). In contrast, late selection theorists argue that words are processed relatively automatically requiring few processing resources such as spatial attention (Allport, 1977). Rather than an all or nothing approach, familiarity-sensitive models posit that identification of familiar words requires fewer attentional resources than unfamiliar words (LaBerge & Brown, 1989; Mozer & Behrmann, 1990). Thus familiarity sensitive models predict that spatial attention is required to process

nonwords and low frequency words but not familiar words (see McCann, Folk, & Johnston, 1992).

According to the dual-route conceptualisation of word recognition, it has been argued that reading via the nonlexical route requires the serial left to right allocation of spatial attention, whereas reading via the lexical route is a parallel process (Cestnick & Coltheart, 1999). Thus, spatial attention deficits would affect phonological coding (sublexical processing) to a greater extent than orthographic coding (lexical processing) (e.g., Cestnick & Coltheart, 1999). Other models have been proposed to account for the role of spatial attention in word recognition within a connectionist framework (Ans et al., 1998; Mozer & Behrmann, 1990). For example, the connectionist multi-trace memory model of polysyllable word reading (Ans et al., 1998) proposes that reading relies on global and analytic attentional mechanisms for processing familiar and unfamiliar words respectively. Pivotal to this model, is a left-to-right moving visual attentional window (VAW) that extends the size of the whole letter string during global processing and is narrowed during analytical processing. Other models of word recognition posit that parietal spatial attention mechanisms act to gate the flow of information to word recognition areas in the ventral stream during reading (LaBerge & Brown, 1989; Mozer & Behrmann, 1990), such that information at attended and unattended regions is facilitated and suppressed respectively. In the case of familiar words, pathway strength acts to provide top down activation to assist early selection mechanisms in the accurate identification of words (Mozer & Behrmann, 1990). However, the identification of unfamiliar or nonwords relies more heavily on early selection processes.

Consistent with familiarity sensitive models of word recognition, a brief visual cue near the beginning or end of letter strings affects naming accuracy of centrally presented unpronounceable nonwords to a greater extent than pseudowords and real words respectively (Auclair & Sieroff, 2002; Sieroff & Posner, 1988). Further, word length effects are greater for nonwords relative to real words and an increase in word naming latency is observed when

phonemes are represented by multiple letters than when they have a direct one to one correspondence (the ‘whammy effect’) (Rastle & Coltheart, 1998).

Patients with RH parietal lesions typically show greater neglect for pseudowords relative to real words (Brunn & Farah, 1991; Sieroff, Pollatsek, & Posner, 1988). Similarly, Mayall et al. (2001) found that mixed casing increased activation in RH parietal areas for words but not for pseudowords or consonant strings. It was suggested that mix-casing disrupts the normal automatic processing of words and results in facilitation of parallel feature processing or the serial allocation of attention to letters. The word length effect is also greater in the LVF (Ellis, Young, & Anderson, 1988; Whitney & Lavidor, 2004) further implicating activation of the RH in the serial processing of words during reading.

These findings are consistent with the notion that dyslexia is associated with a RH attentional deficit that affects the reading of nonwords or unfamiliar words which require phonological decoding to a greater extent than real words. However, few studies have investigated the link between phonological decoding and spatial attention in dyslexia. As previously mentioned, Facoetti et al. (2006) recently found a reduction in the inhibition effect in the RVF among a sub-group of dyslexics with a nonword reading deficit. The reduction in the RVF inhibition effect correlated significantly with nonword reading accuracy ($r = 0.55$) and accounted for unique variance (26.1%) in nonword but not real word reading after accounting for age and IQ. RVF attentional inhibition also contributed unique variance (17.2%) to nonword reading accuracy after accounting for phonological skill (phoneme blending), suggesting that it contributes to nonword reading independently of auditory-phonological mechanisms. The findings of Kinsey et al. (2004) also suggest a stronger association between spatial cueing effects and nonword reading than irregular word reading. Some findings suggest that spatial attention difficulties affect orthographic (visual) processing of words to a greater extent than phonological (auditory) processing (Thomson et al., 2005). However, there is also evidence to suggest that the spatial attention deficits observed among dyslexics occur in both the visual and auditory

modality (Facoetti et al., 2005; Hari & Renvall, 2001; Vidyasagar, 1999) which is consistent with a multi-modal attentional deficit and may account for variance in both orthographic and phonological ability.

Vidyasagar has proposed a model based on the familiarity sensitive model of LaBerge and Brown (1989) which attempts to account for spatial attention difficulties observed among dyslexics in terms of underlying neuroanatomical attentional networks (see Vidyasagar, 1999, 2001, 2004; Vidyasagar & Pammer, 1999). According to this theory, damage to the M system, or areas of the PPC, disrupts the attentional spotlighting mechanism in dyslexia. As previously mentioned incoming visual information is modulated by attention as early as the primary visual cortex in both Macaque monkeys (Vidyasagar, 1998) and humans (Martinez et al., 1999; Martinez et al., 2001). According to Vidyasagar (2004), areas of the dorsal visual stream provide feedback projections that act to gate the flow of information to object identification areas in the ventral stream. The attentional feedback is thought to originate in the PPC, which receives largely M input and acts as a spotlight to select relevant locations for subsequent identification in extra-striate areas of the ventral stream. During reading, this attentional spotlight mechanism is thought to shift the focus of attention to allow processing of one or two letters at a time. Thus, learning to read requires the ability to train the spotlight mechanism to perform spatially sequential serial searches. Vidyasagar suggests that a visuo-spatial attention deficit could affect both the orthographic (lexical) and phonological (sublexical) routes to reading. However, it is possible that different subtypes of dyslexia reflect dysfunction in different areas of the magnocellular or dorsal visual processing stream, or the development of different strategies to compensate for an attentional deficit (Vidyasagar, 2004).

This theory is consistent with findings that sensitivity to the position of letters in word strings predicts reading ability and word recognition skills in both children and adults (Pammer, Lavis, Cooper, Hansen, & Cornelissen, 2005; Pammer, Lavis, & Cornelissen, 2004). On the basis of findings that a spatial encoding task (indexing letter position encoding) and the M-

mediated frequency doubling illusion made independent contributions to reading ability, Pammer, Lavis, and Cornelissen (2004) proposed the existence of two visual encoding mechanisms that are functionally distinct: One fine scale mechanism important for the spatial discrimination of letters within words and the other a coarse scale spotlighting mechanism involved in the spatial localisation of words within text. This theory is also consistent with research showing that an intervention aimed at training dyslexics to attend to grapheme position resulted in improved phonological decoding and improved reading comprehension and phonological awareness (McCandliss, Beck, Sandak, & Perfetti, 2003). However, Roach and Hogben (2004) have also shown that spatial cueing deficits may be present independently of impairment in the magnocellular system as measured by flicker sensitivity and motion sensitivity.

Summary

Numerous lines of evidence indicate that dyslexics differ from normal readers on tasks that assess the functioning of spatial attention. Dyslexics take longer to search for feature conjunctions and show a greater increase in search times as a function of distractor set size, suggesting a deficient attentional spotlighting mechanism (Vidyasagar & Pammer, 1999). The findings of covert orienting studies suggest that dyslexia is associated with a deficit in automatic orienting (Facoetti et al., 2000b), as well as differences in hemispheric control (Facoetti et al., 2001) and inhibitory processing. It has also been argued that dyslexics have a diffuse focus of attention and have difficulty in narrowing or focusing attention. The process of attentional capture is slower in dyslexia and they may also have difficulties disengaging attention and inhibiting stimuli that are not the current focus of attention (Hari & Renvall, 2001). Several lines of converging evidence indicate differences in the hemispheric control of attention in dyslexia which manifest as LVF mini-neglect and over-distractibility in the RVF (Hari et al., 2001). Together these findings have been interpreted to occur due to a RH parietal deficit which may be

related to the functioning of the magnocellular visual processing stream (Facoetti et al., 2003a; Facoetti & Turatto, 2000; Facoetti et al., 2001; Hari & Renvall, 2001; Hari et al., 2001; Vidyasagar & Pammer, 1999). There is also some evidence to suggest that attentional difficulties extend to the auditory system (Facoetti et al., 2005; Facoetti et al., 2003b) which further implicates multimodal areas of the PPC. Few studies have examined the relationship between phonological decoding and spatial attention. However, familiarity sensitive models of reading (e.g., LaBerge & Brown, 1989) predict that spatial attention is particularly important for the sequential analysis of graphemes during phonological decoding. According to Vidyasagar (1999), dyslexia stems from a deficient spotlighting mechanism that originates in the PPC and affects subsequent processing in the ventral visual stream. A large body of electrophysiological research has shown that selective visual attention to spatial locations modulates the early visual components of the ERP waveform (Mangun, 1995). However, few electrophysiological studies have directly investigated the link between spatial attention and reading disability. The following chapter aims to review the findings of electrophysiological research in dyslexia with particular focus on research that has investigated indices of early visual and attentional processing.

CHAPTER 4 – ELECTROPHYSIOLOGY, ATTENTION AND DYSLEXIA

Event-Related Potentials (ERPs)

The present chapter outlines the neurobiological correlates of reading disability drawing on findings from electrophysiological research. The electroencephalogram (EEG) measures voltage fluctuations recorded from electrodes on the scalp. ERPs are the average of the brain's response to multiple presentations of a given stimulus (for reviews see Fabiani, Gratton, & Coles, 2000; Key, Dove, & Maguire, 2005). ERPs have a fine temporal resolution allowing for the mapping of momentary changes in brain activation and the investigation of the time course of cognitive processes. Although functional localisation of cognitive processing is best investigated with the fine spatial resolution offered by neuroimaging techniques, recent advances in source localisation techniques have allowed for localisation of the generators of ERP components (Key et al., 2005).

ERP waveforms are typically described in terms of positive and negative going deflections and are conventionally labelled according to polarity and latency (e.g., N1 is a negative deflection occurring approximately 100ms after stimulus onset). ERP components are associated with a range of cognitive processes including expectancy (CNV), sensory experience (P1), selective attention (N1), active discrimination of stimulus features (N2), delivery of task relevant information (P300 or P3) and semantic processing (N400) (Key et al., 2005). The early components of the ERP waveform (e.g., P1, N1) are sensitive to physical characteristics of stimuli and are termed exogenous, whereas the later components (e.g. N4, P3) are sensitive to information processing aspects of stimulus processing are termed endogenous (Donchin, 1978). Several components (e.g., N2, P2) are sensitive to both physical and information processing aspects of stimuli and are referred to as transient (Hugdahl, 1995) or mesogenous (Fabiani et al., 2000). A brief description of the main exogenous, mesogenous, and endogenous ERP components is provided below. However, an extensive discussion of the temporal properties,

localisation, and functionality of ERP components is beyond the scope of the present review and can be found elsewhere (see Fabiani et al., 2000; Hugdahl, 1995; Key et al., 2005).

Exogenous Components.

The occipital P1 component is elicited approximately 100ms after the onset of a visual stimulus and has been localised in ventral and lateral occipital regions, suggesting a striate or extra-striate (posterior fusiform gyrus) origin (Key et al., 2005). P1 amplitude is greater at RH than LH posterior sites for passive viewing, thickened letter detection, case mismatch detection, and lexical decision suggesting that it reflects visual analysis common to the perception of any visual input (Compton, Grossenbacher, Posner, & Tucker, 1991).

The N1 component consists of three sub-components: the anterior N1 (~140ms), an occipito-parietal component (150-160ms), and an occipito-temporal component (170-200ms) (Clark & Hillyard, 1996; Johannes, Münte, Heinze, & Mangun, 1995). The anterior component may be related to overlapping responses and preparatory activity as it decreases when SOA is decreased or when the task does not require a motor response (Vogel & Luck, 2000). In contrast the posterior N1 components are typically elicited during stimulus discrimination tasks and are thought to be related to selective attention (Vogel & Luck, 2000).

Both posterior P1 and N1 ERP components are modulated by attention to location, such that greater amplitude is observed when stimuli appear at attended relative to unattended locations (for reviews see Eimer, 1998; Mangun, 1995). Rather than reflecting attentional processes (e.g., orienting, disengaging or engaging) it has been argued that these attention effects reflect the modulation of visual processing as a result of these mechanisms (Luck, 1995), most likely in the form of a 'sensory gain control' or amplification mechanism which acts to decrease signal to noise ratio and facilitate perceptual processing of attended locations in extrastriate areas (Clark & Hillyard, 1996; Hillyard, Vogel, & Luck, 1998; Hopf, Vogel, Woodman, Heinze, & Luck, 2002; Mangun, 1995; Mangun et al., 2001). This attentional modulation is thought to act

like a spotlight to facilitate processing of information within bounds the current attentional focus (Mangun et al., 2001).

There is some evidence that the P1 and N1 attention effects represent dissociable processes that reflect the suppression of information at unattended locations and the enhancement of processing at attended locations respectively (Luck et al., 1994). For example, in a covert orienting task, Luck et al. (1994) found greater P1 amplitude for invalid relative to valid trials and greater N1 amplitude for valid relative to invalid trials. Mangun (1995) tentatively argued that the P1 component and the occipital-temporal N1 reflect processing within the ventral pathway, whereas the parietal N1 component is related to processing within the dorsal visual pathway. However the findings of some source localisation studies do not necessarily support this hypothesis (Wijers et al., 1993). Further, the N1 discrimination effect is the same for tasks subserved by the ventral (colour) and dorsal (motion) visual pathways suggesting that it represents the process of discrimination within the focus of spatial attention that is not necessarily related to the “what/where” distinction (Vogel & Luck, 2000).

Orthographic analysis has also been associated with early occipito-temporal negativity within the N1 time frame, possibly reflecting the activation of the visual word form area. Greater left lateralised N125 amplitude has been shown for consonant strings in comparison to words (Compton et al., 1991), and a left lateralised occipito-temporal N170 component has been shown to index orthographic discrimination and possibly visual word form processing (Bentin, Mouchetant-Rostaing, Giard, Echallier, & Pernier, 1999; Simon, Bernard, Largy, Lalonde, & Rebai, 2004). Bentin et al. (1999) found greater N170 amplitude at temporal sites in the LH for orthographic stimuli and the RH for non-orthographic stimuli in a visual discrimination task. Simon et al. (2003) found greater N170 for letter stimuli than pseudo-letters at LH temporal sites and greater N170 for pseudo-letters than consonant strings in the RH. In summary, these findings suggest hemispheric differences in early visual processing that differ as a function of the lexical status of the stimulus.

Mesogenous components

The visual P2 or P200 is a positive component elicited approximately 150-200ms post-stimulus at frontal sites, and 200ms post-stimulus at occipital sites (Key et al., 2005). Source localisation studies have implicated generators in bilateral inferior occipital (extra-striate) areas. The P2 component is elicited by a range of tasks including selective attention, feature detection, early sensory encoding, stimulus change, and short term memory, and its amplitude is modulated by stimulus complexity (see Key et al., 2005).

Modulation of the N2 or N200 is often associated with the detection of a mismatch between the features of the stimulus and/or between a previously formed template and the current stimulus, particularly at frontal sites (Fabiani et al., 2000). A frontal N2 component (100-300ms) is elicited on nogo trials in go/nogo paradigms, and is thought to be associated with response inhibition that is independent of motor output processes (Kopp, Mattler, Goertz, & Rist, 1996a). Studies employing flanker paradigms have found greater N2 amplitude for incompatible in comparison to compatible flanker stimuli further suggesting that it indexes inhibitory control (Heil, Osman, Wiegelmann, Rolke, & Hennighausen, 2000; Kopp et al., 1996a; Kopp, Rist, & Mattler, 1996b).

An N2 component (200-400ms) with a central maximum is related to the processing of task relevant stimuli and is thus often termed selection negativity. The visual N2 is typically maximal over pre-occipital electrodes and different distributions have been identified for letter strings, complex pictures, and faces, indicating that it may reflect category-specific processing (Key et al., 2005). However, there is some ambiguity in the literature regarding the distinction between the N200 component and the N170 component mentioned above. For example, selection negativity may occur as early as 140-180ms (Hillyard & Anllo-Vento, 1998).

Endogenous Components

The P3 or P300 component has been widely studied in relation to a number of cognitive processes. The P300 varies as a function of task relevance and stimulus probability and is thought to index context updating in working memory (e.g., Donchin & Isreal, 1980), cognitive resource allocation (e.g., Kramer, 1991), and other higher order processes such as stimulus evaluation and categorisation (for a review see Key et al., 2005). A distinction has been made between the P3b component which is maximal at central parietal sites and the P3a component that is maximal at fronto-central sites (Comerchero & Polich, 1999). The P3b component elicited by low probability target stimuli is argued to index the allocation of central resources that are relatively independent of the processes of motor preparation and execution (Kok, 1997). In contrast, the P3a component elicited by low probability novel or distractor stimulus is shorter in latency and may index a relatively automatic attentional switching mechanism that is associated with orienting towards novel and biologically relevant stimuli (Comerchero & Polich, 1999).

The N400 is elicited during linguistic tasks and is thought to index lexical integration and semantic processing (Kutas & Hillyard, 1980; Nobre & McCarthy, 1995). For example, Kutas and Hillyard (1980) found that words at the end of a sentence that were not consistent with the meaning of the sentence produced an enhanced N400 component. The N400 component has also been found to be sensitive to phonological and orthographic congruity, and a reduction in N400 has been shown for target words that are preceded by semantically related primes (Miles & Stelmack, 1994).

Electrophysiology and Reading Impairment

The following section reviews the large body of electrophysiological research that has investigated visual ERPs in dyslexia (for other reviews see Breznitz, Shaul, & Gordon, 2003). A review of electrophysiological research investigating auditory processing in dyslexic and language impaired individuals is beyond the scope of the present review and can be found

elsewhere (see Leppänen & Lyytinen, 1997; Lyytinen et al., 2005). Research in this area can be broadly classified into studies that have investigated low level visual processing, selective attention, spatial attention, and linguistic processing. Research findings in relation to the P200, N200 and later endogenous components such as the P300 and N400 are reviewed briefly, but early visual processing and therefore the exogenous or sensory components (N1, P1) of the ERP waveform are of particular relevance to the present research. Some VEP studies refer to the negative component occurring at approximately 50-70ms as an N1 component. In the present thesis this component is referred to as the C1 and reserves the term N1 to refer to the first negative peak occurring after the visual P1 component.

Low level visual processing – Visual Evoked Potentials (VEPs)

Several studies have investigated the possibility that dyslexia stems from a low level visual processing deficit by examining early visual ERP components (e.g., C1, P1 and N1) and ERP complexes (e.g., C1-P1, P1-N1). Some of these studies have provided support for the M hypothesis by showing differences in VEPs between dyslexics and control subjects for stimuli of low relative to high contrast, spatial frequency, or luminance (Lehmkühle et al., 1993; Livingstone et al., 1991; May et al., 1991; Romani et al., 2001) (but see Breckel, Struelens, & Raic, 1996; Farrag, Khedr, & Abel-Naser, 2002; Johannes, Kussmaul, Münte, & Mangun, 1996; Victor, Conte, Burton, & Nass, 1993). Further support for an M deficit in dyslexia comes from studies finding differences in motion onset potentials such that dyslexics show greater differences in early ERP components for moving relative to stationary stimuli (Kubova, Kuba, Peregrin, & Novakova, 1995; Schulte-Körne, Bartling, Deimel, & Remschmidt, 2004).

Several passive VEPs studies have shown that dyslexics show a reduction in P1 amplitude in response to both stationary (Solan, Sutija, & Ficarra, 1990) and moving stimuli (Schulte-Körne et al., 2004) and increased P1 latency to low but not high contrast checkerboard stimuli (Livingstone et al., 1991), and low but not high frequency stimuli (Lehmkühle et al.,

1993) (but see Brecelj et al., 1996; Farrag et al., 2002). However, other authors have reported no differences in P1 amplitude (Johannes et al., 1996; Schulte-Korne, Bartling, Deimel, & Remschmidt, 1999; Victor et al., 1993) or P1 latency (Brannan, Solan, Ficarra, & Ong, 1998; Johannes et al., 1996; Kubova et al., 1995; Victor et al., 1993) between dyslexics and controls in response to checkerboard patterns or grating stimuli.

Most of the studies mentioned so far have recorded VEPs from midline occipital and central sites, however, differences in the laterality of ERP components have been investigated by some researchers. For example, Mecacci et al. (1983) found that male dyslexics ($n=16$) showed lower N1-P2 amplitude in comparison to male controls ($n=8$) in response to checkerboard stimuli of varying spatial frequencies. Furthermore, whereas controls showed a symmetrical hemispheric distribution of this component, only four dyslexics showed symmetry, with the remainder showing either greater amplitude in the LH ($n=5$), or the RH ($n=7$). The relationship between spatial frequency and amplitude was significant for symmetrical dyslexics, not for LH asymmetrical dyslexics and only in the RH of right asymmetrical dyslexics. Schulte-Korne et al. (1999) found that controls but not dyslexics showed greater P1 and P2 amplitude in the RH for low (2 c/deg) in comparison to high (11.33 c/deg) spatial frequency stimuli. In response to low spatial frequency checkerboard stimuli, Hennighausen et al. (1994) found that an early negative potential (160-190ms) at central LH electrodes was present in 44% of male dyslexics and 80% of controls, and was particularly reduced in those dyslexics with a greater spelling score/IQ deviance.

Selective Attention

Several ERP components (e.g., N1, N2, and P3) are associated with the selection of visual stimuli for cognitive processing. As previously noted, the N1 component is sensitive to the selection of relevant locations, whereas the N2 and P3 components are enhanced for target stimuli possessing task relevant features and the P2 component is thought to index perceptual

processing or stimulus discrimination. Studies that have investigated selective attention among reading disabled populations have employed passive viewing paradigms in which no responses were required as well as selective paradigms in which responses are required to either low probability (oddball paradigm) or equi-probable (go/nogo paradigm) targets. However, these studies vary considerably in terms of the paradigm used, the selection and characteristics of subjects, and the measurement and analysis of ERP components. For example, some studies analysed ERPs to targets or go stimuli, while others analysed ERPs to standard or nogo stimuli or difference waveforms in which the ERPs of standard were subtracted from target stimuli. Although it is difficult to compare these studies directly, the following section briefly summarises some of the more common findings with particular attention to studies that have investigated early visual processing and hemispheric differences.

During the performance of selective attention tasks, dyslexic or reading impaired participants often show a reduction in P300 amplitude and an increase in P300 latency relative to controls (Breznitz, 2002; Duncan et al., 1994; Harter, Anllo-Vento, Wood, & Schroeder, 1988a; Harter, Diering, & Wood, 1988b; Holcomb, Ackerman, & Dykman, 1985; Naylor, Wood, & Harter, 1995; Taylor & Keenan, 1990). In contrast, Russeler et al. (2003) found that male adult dyslexics selected on the basis of spelling ability showed greater P300 amplitude to low probability target stimuli under passive conditions and greater frontal P3 amplitude for nogo trials suggesting difficulty allocating attentional resources. However, considering that there were no behavioural differences, it is possible that the increase in P300 amplitude was related to compensatory processing among the adult dyslexics in this study. Whereas there is some evidence that ADHD symptoms contribute to the P300 effects observed among dyslexics (Duncan et al., 1994; Holcomb et al., 1985), other research findings suggest independent sources of P300 reduction for participants with concomitant ADHD and dyslexia (Harter et al., 1988a; Harter et al., 1988b).

Some researchers have found that dyslexics show increased N200 latency relative to controls during the performance of selective attention tasks. For example, Taylor and Keenan (1990) found longer latency N200 (target minus non-target) among dyslexic children during linguistic and non-linguistic visual oddball tasks. Duncan et al. (1994) found increased N200 latency (target minus standard letters) among dyslexics relative to controls for a simple reaction time but not go/nogo, or choice reaction time oddball tasks. Similarly, Neville et al. (1993) found increased N230 latency among language impaired individuals with a concurrent reading disability to visual standards in an oddball task. However, Harter et al. (1988) found no effects of group or relevance on the N230 component in a selective attention task with equi-probable stimuli. Neville et al. (1995) found that dyslexic adults showed greater N290 amplitude in comparison to both normal readers and borderline reading impaired adults. However, it is possible that this effect was partly attributable to the reduction in positivity observed among dyslexics in this study. Russeler et al. (2003) found no differences between adult dyslexics or controls in the amplitude, latency, or laterality of the fronto-central N2 component to nogo stimuli indicating intact inhibitory processing but there were no behavioural differences between dyslexics and controls in this study.

Differences in P2 amplitude have also been reported within the selective attention literature, with several studies indicating greater differences in the language dominant LH. For example, Harter et al. (1988b) found that children with reading disability showed smaller P240 amplitude at LH central sites, whereas controls showed greater amplitudes in the LH relative to the RH, consistent with a LH linguistic deficit (Harter et al., 1988b). Naylor et al. (1995) also found a bilateral reduction in the P240 component using a selective attention paradigm requiring a response to letter stimuli among geometric patterns. Although the reduction was bilateral, P240 amplitude in the LH but not RH correlated with reading ability. Preston, Guthrie, Kirsch, Gertman, and Childs (1977) investigated ERPs of dyslexic ($n=9$) adults and controls ($n=9$) under either passive viewing (light flash oddball) or silent counting of target words. Increased P2

amplitude was found in the LH for words relative to light flash stimuli, and this difference was greater for controls than dyslexics. Increased P200 latency has been observed among dyslexic children during the performance of both linguistic and non-linguistic selective attention tasks (Breznitz, 2002), while other studies have found increased P200 latency for linguistic tasks but not non-linguistic tasks (Breznitz & Meyler, 2003) or no differences in P200 latency (Breznitz & Meyler, 2003) among dyslexic adults.

Few of the selective attention studies mentioned so far have investigated the early exogenous components of the ERP waveform. Neville et al. (1993) reported a reduction in occipital P150 amplitude to standard stimuli, regardless of rates of stimulation or whether stimuli were presented centrally or peripherally, in language impaired individuals with a concurrent reading disability. An increase in N150 latency was also observed at frontal and anterior temporal sites, as well as a reduction in N150 amplitude for centrally presented stimuli. Naylor et al. (1995) found a reduction in P150 amplitude for reading disabled ($n=10$) compared to non-reading disabled ($n=8$), and borderline reading disabled ($n=14$) adult males in a selective attention task which required a response to letter stimuli among geometric patterns. However, this effect was only a trend after controlling for IQ and there were no differences in the behavioural data. Other studies have found no differences in the P1 (Harter et al., 1988a) or N1 (Duncan et al., 1994; Lovrich, Cheng, & Velting, 2003) components during selective attention tasks.

Some early research findings are consistent with differences in early processing in the language dominant LH. For example, using a task in which low probability light flashes were detected from bright light flashes, Conners (1970) found a reduction in amplitude for early negativity (140-200) to standard stimuli at left parietal sites for relatively poor readers in comparison to relatively good readers with a concurrent learning disorder. In a passive viewing study, Preston et al. (1974) reported greater N180 amplitude to word stimuli in comparison to light flashes, and an overall reduction in N180 at LH parietal sites (P3) for reading disabled

($n=9$) relative to chronological age matched ($n=9$) and reading age matched ($n=9$) controls. In a study in which words were passively viewed, dyslexics showed a negative peak at about 175ms, whereas controls showed more complex waveforms with both an earlier (~120ms) and a later (~195ms) negative peak observed prior to 200ms (Symann-Louett, Gascon, Matsumiya, & Lombroso, 1977). Similarly, Cohen and Breslin (1984) measured VEPs in response to white flashes or words in dyslexic ($n=16$) and normally reading male children. For word stimuli, control children showed lower cross correlations between the RH and LH compared to dyslexics for N1 latency, indicating greater hemispheric specialisation. Cross correlations within each hemisphere also indicated greater specialisation in the LH for normal readers. However, this study did not report analyses of mean amplitude and latency of the N1 component.

Spatial Attention and Covert Orienting

Few researchers have investigated the electrophysiological correlates of spatial attention in relation to reading ability (Harter et al., 1989; Jonkman et al., 1992; Licht, Jonkman, Bakker, & Woestenburg, 1990; Wijers, Been, & Romkes, 2005). While some of these have investigated covert orienting, none have employed the same paradigm and each differs in terms of the ERP timeframes investigated and the analyses performed on the data making direct comparison difficult. In addition, none of these studies have specifically investigated the modulation of the early exogenous components (P1, N1) of the ERP waveform by spatial attention.

Some studies have used sustained orienting paradigms in which targets preceded by symbolic spatial cues require a response only if presented in the attended visual field. For example, Wijers, Been, and Romkes (2005) measured ERPs in the cue-to-target interval (750ms) as a measure of the executive attentional control in adult dyslexics ($n=11$) and controls ($n=11$). Dyslexics were slower to respond to target stimuli and tended to be less accurate in comparison to controls. Controls showed greater positivity in the RH (~350ms) when attention was sustained to the LVF. This effect was significant in both the LH and RH in dyslexics, possibly indicating

anomalous lateralisation of prefrontal attentional control processes. Licht et al. (1990) found that both P-type and L-type dyslexics were slower than normal readers and that L-type were less accurate than P-type dyslexics. L-type dyslexics showed greater negativity over frontal sites and P-type dyslexics showed greater positivity over occipital sites within the 110-200ms epoch post target stimulus, suggesting a possible distinction between the reliance of anterior and posterior attentional networks. However, these findings should be interpreted with caution due to a lack of methodological detail and statistical analyses reported by the authors.

Using a trial-by-trial cuing location paradigm in which responses were required to targets preceded by valid spatial cues and withheld for stimuli preceded by invalid spatial cues, Jonkman, Licht, Bakker, and Van den Broek-Sandmann (1992) found that P-type ($n=21$) and L-type ($n=22$) dyslexics were slower to respond and less accurate in comparison to normally reading ($n=22$) Dutch children. Within 100-200ms post target stimulus presentation, normal readers showed larger positivity for invalid than valid trials, P-type dyslexics showed greater positivity for valid in comparison to invalid trials, and L-type dyslexics showed greater positivity for LVF in comparison to RVF trials overall. Harter, Anllo-Vento, and Wood (1989) investigated the effects of uninformative central spatial cues on ERPs in male dyslexic ($n=12$) and control ($n=15$) children diagnosed with or without concurrent ADHD. There were no group differences in behavioural data. However, N1 amplitude (fixed latency 200ms) in response to relevant stimuli was greater for dyslexics in comparison to controls, particularly in the RH for LVF trials. These findings were interpreted to indicate a reduction in inter-hemispheric competition or inhibition due to a lesion in the LH, or enhanced spatial attention or visuo-spatial strength in peripheral vision, possibly reflecting compensation for reduced later non-spatial target selection as indexed by a reduction in P300 amplitude in the LH.

Linguistic Processing

Most of the studies that have investigated the electrophysiological correlates of dyslexia during linguistic processing have investigated the later endogenous components of the ERP waveform. A common finding is reduced amplitude and increased latency of P2, P3, and N4 components among dyslexics, particularly in the LH. Research that has investigated linguistic processing in dyslexia using priming, sentence reading, and lexical decision paradigms are reviewed briefly below with particular focus on those studies that have also investigated the earlier sensory/perceptual components of the ERP waveform.

Priming paradigms. Modulation of the N400 component is found during the performance of priming tasks that involve word recognition such that there is a reduction in N400 amplitude for previously primed words. Reading disabled children show a reduction in N400 amplitude to unprimed words relative to control children despite normal N400 priming effects (Stelmack & Miles, 1990; Stelmack, Saxe, Noldy-Cullum, Campbell, & Armitage, 1988), suggesting intact short term memory but a failure of semantic access in long term memory (Stelmack & Miles, 1990). However, in another study Miles and Stelmack (1994) found evidence for abnormal priming among reading disabled children, such that there was a lack of N400 amplitude reduction for words previously primed by both spoken words and pictures (Miles & Stelmack, 1994). These findings were interpreted as reflecting a deficit in auditory-verbal and visual-spatial processing during associative processing. Normal readers but not reading disabled children showed greater frontal N450 in the LH in comparison to the RH suggestive of greater lateralisation of language function.

Other researchers have investigated ERPs in reading disabled populations during phonological priming tasks (Ackerman, Dykman, & Oglesby, 1994; McPherson, Ackerman, Holcomb, & Dykman, 1998; McPherson, Ackerman, Oglesby, & Dykman, 1996). Whereas normal participants show a reduction in N400 amplitude to target words that are previously

primed by a word with similar orthography or phonology, a reduction in the N400 rhyming effect has been found for 'dysphonetic' dyslexics with poor nonword reading scores but not 'phonetic' dyslexics with better nonword reading scores (McPherson et al., 1996). However, no control group was included in this study. McPherson et al. (1998) found that dysphonetics showed reduced phonological priming but normal orthographic priming, whereas phonetics showed both phonological and orthographic priming.

Together these findings are consistent with a deficit in linguistic processing, however, few priming studies have examined the earlier sensory/perceptual components (e.g., P1, N1) of the ERP waveform and others have yielded inconsistent findings. For example, Stelmack et al. (1988) found that dyslexics showed greater N1 amplitude at lateral temporal sites relative to controls during the acquisition phase of a recognition memory priming task. In contrast, Stelmack and Miles (1990) found no group differences for frontal N150 amplitude or latency. However, dyslexics showed greater occipital P160 amplitude to unprimed words. McPherson et al. (1996) found that phonetics showed greater N1 amplitude at RH parietal sites suggestive of greater use of attentional resources for phonetic processing. In contrast, dysphonetics showed greater N1 amplitude in the left hemisphere. However, few behavioural differences were observed in this study.

Sentence reading tasks. The amplitude of the N400 component is also greater for sentences ending in incongruous than congruous sentences suggesting that it reflects the integration of words into meaningful context. Greater N400 amplitude to both congruous and incongruous sentence endings have been observed among both language impaired children (Neville, Coffey, Holcomb, & Tallal, 1993) and dyslexic adults (Robichon, Besson, & Habib, 2002), suggesting difficulty in integrating words into the context of the sentence. However, in one study dyslexic children showed a reduction in N400 amplitude for incorrect sentence endings relative to controls (Brandeis, Vitacco, & Steinhausen, 1994).

Neville et al. (1993) also found that language impaired individuals showed a reduction in P150 amplitude relative to controls. Similarly, Brandeis et al. (1994) found that dyslexic children tended to show a reduction in P110 amplitude in the LH, and shorter P110 latency overall in comparison to control children. P1 amplitude tended to correlate with reading ability for dyslexics but not controls. However, the significance level for these comparisons was $p < 0.1$. Robichon, Besson, and Habib (2002) did not investigate the P1 and N1 components directly but found no difference between dyslexic adults and controls in the amplitude of the overall N1-P2 complex which is thought to sensory perceptual analysis of physical characteristics.

Lexical decision tasks. Breznitz and colleagues have published a number of studies that have investigated the electrophysiological correlates of lexical decisions among reading disabled populations. In these tasks participants were required to decide whether homograph or homophone pairs looked the same (orthographic) or sounded the same (phonological) respectively (Breznitz, 2002, 2003), or to decide whether word pairs were real words (orthographic) or pseudowords (phonological) (Breznitz & Misra, 2003; Miller-Shaul & Breznitz, 2004). A further rhyming decision task was used in some studies in which participants were required to decide whether word pairs rhymed or not (phonological) (e.g., Breznitz, 2002, 2003).

Both male dyslexic children (Breznitz, 2002; Miller-Shaul & Breznitz, 2004) and compensated adults (Breznitz, 2003; Breznitz & Misra, 2003; Miller-Shaul & Breznitz, 2004) show longer reaction times and longer P2 and P3 latencies during lexical decision tasks and these latency differences are typically greater for decision tasks that require phonological relative to orthographic processing (Breznitz, 2002, 2003; Miller-Shaul & Breznitz, 2004). Another common finding is that the speed of processing gap between auditory/phonological and visual/orthographic decision tasks is greater for dyslexics relative to controls (e.g., Breznitz, 2002, 2003; Breznitz & Misra, 2003). The increased time required to make phonological relative

to orthographic decisions was thought to relate to the sequential versus holistic nature of these processing systems, and is consistent with an increase in the demands on cognitive resources during phonological processing (Breznitz, 2003). It has been argued that this speed of processing asynchrony affects the cross-modal integration of grapheme and phoneme information which may explain the temporal processing abnormalities and decreased reading speeds observed in dyslexia (see Breznitz, 2005). Differences in speed of processing asynchrony between adults and children suggest that decoding problems stem from low level perceptual and attentional difficulties (P200) which may in turn affect later memory processes (P300) (Breznitz & Berman, 2003).

Few studies employing lexical decision tasks have reported findings with regard to early exogenous components of the ERP waveform. Breznitz (2003) found no group differences in the amplitude or latency of the N1 component for linguistic go/nogo tasks, but dyslexics showed greater N1 amplitude in comparison to controls for homophone pairs during a phonological/orthographic decision task. Breznitz (2005) found that dyslexics showed delayed N1 latency for letter but not object naming tasks. Further, Wimmer, Hutzler, and Wiener (2002) found a reduction of N1 amplitude (50-150ms) among dyslexic children for pseudowords but not real words during a word naming task, as well as a reduction in N1 amplitude at LH frontal sites regardless of word type. The reduction observed in the RH was thought to be consistent with a deficit in attentional processes associated with RH parietal areas as reading pseudowords requires the use of a sequential attentional strategy. However, this task did not purposefully manipulate spatial attention. The use of a strategy to limit attention to a smaller number of letters was also considered as a possible explanation for these findings.

Hemispheric Asymmetries and Electrophysiology

From the literature reviewed so far several authors have reported differences between reading impaired and normal readers that are consistent with processing differences in the

language dominant LH. For example, dyslexics show a reduction N1 amplitude in the LH in response to contrast reversal stimuli (Hennighausen, Remschmidt, & Warnke, 1994), during passive viewing of words and light flashes (Conners, 1970; Preston, Guthrie, & Childs, 1974; Symann-Louett et al., 1977). A similar left lateralised reduction in amplitude has been observed for the P100 (Brandeis et al., 1994), P200 (Harter et al., 1988b; Preston et al., 1977), P300, and N400 (Miles & Stelmack, 1994) components of the ERP waveform.

Consistent with the RH hypothesis of dyslexia, the findings of other studies have indicated processing differences between dyslexics and controls at RH sites. For example, controls but not dyslexics showed greater P2 amplitude over the RH occipital cortex in response to stimuli presented at a range of contrasts and spatial frequencies (Schulte-Korne et al., 1999). Further, dyslexics showed a reduction in N100 amplitude in response to nonwords but not words at RH central sites (Wimmer et al., 2002). Wijers et al. (2005) found that controls but not dyslexics showed greater P350 in the RH (~350ms) during the cue-to-target interval when attention was sustained to the LVF.

Interestingly some other research findings are consistent with a reversal in hemispheric asymmetries of some ERP components for dyslexics relative to controls. For example, dyslexics showed a reversal of the normal LH > RH amplitude asymmetry for the P270 and P450 components in form and rhyme letter discrimination tasks respectively (Lovrich et al., 2003). In an S1-S2 paradigm involving language stimuli, the amplitude of the first negative component (230ms) following S1 was greater at LH than RH occipital sites for male control children, while male dyslexics showed a bilateral effect (Jones & Michie, 1986). Furthermore, for non-language stimuli there was a trend for dyslexics to show greater N230 amplitude at RH occipital sites. A similar hemispheric reversal was observed for P340 latency. Barnea, Lamm, Epstein, and Pratt (1994) found that P3 amplitude at central sites was greater in the LH for controls and the RH for dyslexics in a recognition memory paradigm suggesting a reliance on linguistic and visual processes respectively. Landwehrmeyer, Gerling, and Wallesch (1990) found that dyslexics

showed a reduction in negativity in the LH and increased activity in the RH during linguistic tasks. However, it is not clear which component this study is referring to as latency is not mentioned. Although not the focus of this review, some auditory studies have also shown reversals in the hemispheric asymmetry of ERP components (Breznitz & Meyler, 2003; Brunswick & Rippon, 1994; Erez & Pratt, 1992; Mazzotta & Gallai, 1992; Shucard, Cummins, & McGee, 1984). Furthermore, among normal children, the relationship between ERP amplitudes and reading proficiency is greater in the RH for young children and in the LH for older children (Licht, Bakker, Kok, & Bouma, 1988, 1992).

Some research findings indicate that hemispheric differences in electrophysiology are specific to particular subtypes of dyslexia. Whereas it has been suggested that dysphonasia and dysideasia occur due to LH and RH deficits respectively, in an EEG study, it was found that dysideastics and dysphonetics show an over-reliance on the LH and RH respectively which is consistent with a compensation-from-strength model rather than the direction of difference proposed by Boder (Flynn, Deering, Goldstein, & Rahbar, 1992). Consistent with this proposal, McPherson et al. (1996) found that phonetics and dysphonetics showed greater N1 amplitude at RH and LH sites respectively. In further support of the compensatory nature of these differences there were few behavioural differences observed in this study. Other research has shown that stimulating the RH (LVF) in L-type and the LH (RVF) of P-type dyslexia results in a changes in the lateral distribution of ERPs (P250) and reading improvement particularly for L-type dyslexics (Bakker et al., 1981; Bakker & Vinke, 1985). As dysphonetic dyslexics are most similar to L-type dyslexics, these findings are also consistent with the RH deficit hypothesis.

Summary

The electrophysiology of dyslexics differs from that of controls across a range of tasks and ERP components. Differences in the lateralisation of ERP components are generally consistent with a LH linguistic deficit; however, consistent with the RH hypothesis of dyslexia, some research findings indicate processing differences between dyslexics and controls at RH sites as well as evidence for a reversal of hemispheric asymmetries and hemispheric differences between subtypes. Many researchers have not investigated or have not found differences between dyslexics and controls in terms of the early exogenous components of the ERP waveform (P1 and N1). However dyslexics often show reduced amplitude and increased latency of the P1 component during psychophysical, selective attention, and linguistic tasks (Brandeis et al., 1994; Lehmkuhle et al., 1993; Livingstone et al., 1991; Naylor et al., 1995; Neville et al., 1993; Schulte-Korne et al., 2004; Solan et al., 1990). Similarly other research findings have indicated a reduction in the amplitude of the N1 component and increased latency during some of these tasks, particularly in the LH (Breznitz, 2005; Connors, 1970; Hennighausen et al., 1994; Neville et al., 1993; Preston et al., 1974; Symann-Louett et al., 1977). In contrast, some studies have reported greater N1 amplitude among dyslexics relative to controls (Breznitz, 2003; Harter et al., 1989; Stelmack et al., 1988). Although differences have been observed for a number of ERP components during selective and spatial attention tasks, few studies have directly investigated the attentional modulation of the P1 and N1 components in reading disability.

CHAPTER 5 - GENERAL RATIONALE & AIMS

The aim of the present series of experiments was to investigate spatial attention among good and poor adult readers selected on the basis of phonological decoding ability (as assessed by nonword reading). According to the phonological deficit hypothesis, a weak phonological mechanism explains the impairment that dyslexics show on a variety of phonological tasks such as nonword reading as well as deficits observed in rapid naming, verbal learning, and verbal memory (see Bradley & Bryant, 1983; Rack et al., 1992; Stanovich, 1988; Stanovich & Siegel, 1994; Wagner & Torgesen, 1987). Poor phonological awareness persists in adult dyslexics (e.g., Bruck, 1992) and adult compensated dyslexics continue to show nonword reading deficits (Ben-Dror et al., 1991; Bruck, 1990).

Considering the importance of nonword reading or phonological decoding for reading acquisition and subsequent reading ability, samples of undergraduate university students with good and poor phonological decoding skills were recruited for the present research. The use of participants who are enrolled in higher education ensures that reading problems do not reflect low engagement in literary tasks (Bruck, 1990) or current developmental differences. Although the poor phonological decoders recruited in the present research were not necessarily diagnosed with dyslexia in childhood, they are likely to be most similar to those diagnosed with developmental phonological dyslexia (Coltheart et al., 1993) or dysphonasia (Boder, 1971), and depending on impairment on other reading measures, may also be similar to those diagnosed with mixed dyslexia or dysphonasia.

The reading problems experienced in dyslexia are typically thought to result from abnormal cerebral lateralisation and in particular disruption to areas of the LH that are specialised for linguistic processing (Beaton, 1997; Galaburda, 1999; Pugh et al., 2001a). However, dyslexics also experience a number of sensory and perceptual problems and differ from normal readers on a range of tasks that tap functioning of the cerebellar, motor, visual, and auditory systems (Farmer & Klein, 1995; Stein, 2001b; Stein & Talcott, 1999). Several lines of

converging evidence indicate that dyslexics show a deficit in the functioning of the fast transient or magnocellular visual processing pathway (Lovegrove, 1996) and dyslexics with poor phonological skills have been shown to be more impaired on measures of magnocellular visual processing (e.g., Borsting et al., 1996; Ridder et al., 1997; Slaghuis & Ryan, 1999). Dyslexics also differ from controls on tasks that tap the dorsal (parietal) visual processing pathway including the PPC which receives predominantly magnocellular input (Stein & Walsh, 1997).

A growing body of evidence indicates that dyslexics differ from controls in terms of the functioning of spatial attention mechanisms that are subserved by the posterior attention system (Hari & Renvall, 2001; Vidyasagar & Pammer, 1999). For example, dyslexics show impairment on covert orienting, visual search, and flanker tasks and also show evidence for mini-neglect of the LVF. These differences are often interpreted to reflect a RH posterior parietal deficit. According to familiarity sensitive models of attention (LaBerge & Brown, 1989; Mozer & Behrmann, 1990), spatial attention is particularly important for the successive analysis of letters during unfamiliar and nonword reading or phonological decoding. There is also some empirical evidence to suggest attentional difficulties are associated with a nonword reading deficit in developmental dyslexia (Facoetti et al., 2006).

Although many electrophysiological studies have reported early perceptual processing differences in dyslexia as indexed by the P1 and N1 components of the ERP waveform (see Eimer, 1998; Mangun, 1995), few studies have attempted to link spatial attention deficits with the attentional modulation of these ERP components. Accordingly, a major aim of this research is to investigate both the behavioural and electrophysiological aspects of spatial attention among good and poor phonological decoders. Of particular interest is the attentional modulation of ERPs during tasks that assess facilitatory and inhibitory attentional mechanisms and their relation to processing asymmetries in the left and right hemispheres. The following five experiments assess different aspects of spatial attention using a range of paradigms including visual search, global local processing, flanker interference, covert orienting, and lexical decision.

CHAPTER 6 - EXPERIMENT 1: VISUAL SEARCH FOR FEATURE CONJUNCTIONS AND PHONOLOGICAL DECODING ABILITY.

Research using visual search tasks has shown that targets defined by a single salient feature (e.g., colour, form, motion) ‘pop out’ and search times are independent of the number of distractor items in a set, indicating a parallel pre-attentive process. However, if the search target is defined by the conjunction of two feature dimensions such as colour and form (e.g., a red triangle among red squares and green triangles), search time increases linearly with set size, suggesting a serial mechanism. According to feature integration theory (Treisman, 1988; Treisman & Gelade, 1980), feature detectors tuned to dimensions such as motion, colour, and form derive feature maps in parallel and code simultaneously for the same region of visual space through communication with a ‘master map’ of locations. When a single feature is to be detected, this location ‘pops out’ on the master map. In contrast, coding of conjunctions or combinations of features is a serial process in which information from different visual modules or feature maps can only be combined at locations that are currently attended on the ‘master map’. As such, conjunction search requires the serial allocation of spatial attention to each visual item in the display (Treisman, 1988; Treisman & Gelade, 1980).

In more recent formulations of feature integration theory, it has been argued that if a feature is particularly salient, individual feature maps inhibit non-target locations on the master map so that they are not attended during search (Treisman, 1988; Treisman & Sato, 1990). However, according to the guided search model (Wolfe, 1994, 1998; Wolfe et al., 1989), the presence of relevant features in feature maps results in *excitation* for these locations on the master map. This is supported by findings that the search for triple-feature conjunctions is faster than two-feature conjunctions, suggesting that potential target locations receive excitation from target compatible feature maps (e.g., Wolfe et al., 1989). Consistent with these two models, some research findings indicate that both excitatory and inhibitory guidance is used to control search for feature conjunctions (Driver, McLeod, & Dienes, 1992).

In contrast to these early selection theories, attentional engagement theory assumes a late locus of selective attention during visual search (Duncan, 1996; Duncan & Humphreys, 1989). According to this model, features that are consistent with top-down information receive a competitive advantage in terms of neural activation. Separate feature maps with task relevant features are thought to be pre-attentively bound as they share the common feature of location and the rules governing figure-ground segmentation also determine which units in feature maps are bound together to form a perceptual object (Duncan, 1996). Similarity of targets to distractors and distractors to other distractors determines how strongly items are grouped into a perceptual unit and therefore the time taken to resolve competition in favour of the target (see Chelazzi, 1999).

The mechanisms involved in visual search may be similar to those used in the process of reading. For example, the processes that guide excitation of target locations and inhibition of distractor locations may be similar to those that facilitate the identification of attended words or letters and the inhibition of unattended letters or words during reading. Furthermore, both require the rapid integration of information across space (Illes et al., 2000) and the conjoining of stimulus features into perceptual objects. The PPC is involved in the allocation of spatial attention and the search for feature conjunctions (e.g., Ashbridge et al., 1997; Corbetta et al., 1995; Friedman-Hill et al., 1995) and has also been implicated in the aetiology of dyslexia (e.g., Stein & Walsh, 1997).

Research has shown that poor readers are slower than normal readers on visual search tasks requiring serial scanning and/or cancellation of letters (Casco et al., 1998; Williams et al., 1987; Williams et al., 1995). Other studies have investigated the relationship between reading ability and visual search using traditional parallel and serial search paradigms that are less reading-like in nature (Buchholz & McKone, 2004; Casco & Prunetti, 1996; Hayduk et al., 1996; Illes et al., 2000; Ruddock, 1991; Vidyasagar & Pammer, 1999). Whereas some researchers have reported differences between dyslexics and controls for simple feature searches (Hayduk et al.,

1996; Ruddock, 1991), others have found a more shallow function suggestive of a more diffuse mode of attention among dyslexics (Facoetti et al., 2000a). Other findings indicate that dyslexics differ in their performance on conjunction search tasks but not on tasks involving the search for single features (Buchholz & McKone, 2004; Casco & Prunetti, 1996; Illes et al., 2000). Casco and Prunetti (1996) found that dyslexic children showed normal parallel and serial search functions for simple features including conjunctions of colour/orientation, and size/orientation but impaired search functions for multi-featured complex shapes, in which targets and non-targets differed in both identity of features and spatial relationship (K, F), or in their spatial relationship alone (b, p). They suggested that poor readers had difficulty searching for letters and geometric shapes which require the integration of features within a module of the visual system, possibly as a result of reduced efficiency in the parallel process used for locating 'where' in space possible targets exist.

Other studies have demonstrated a steeper search function as a function of distractor set-size for feature conjunction tasks among both dyslexic children (Vidyasagar & Pammer, 1999) and adults with poor phonological processing skills (Buchholz & McKone, 2004). These findings have been interpreted to indicate differences in the functioning of an M-mediated attentional spotlight mechanism (e.g., Vidyasagar & Pammer, 1999). Consistent with this proposal, dyslexics with elevated coherent motion thresholds differ more from controls on conjunction search tasks than those without a concurrent motion coherence deficit (Illes et al., 2000). However, in contrast, Buchholz and McKone (2004) found that performance on visual search tasks and not performance on the M-mediated spatial frequency doubling illusion was related to measures of phonological awareness in adult dyslexics.

The aim of the present study was to investigate visual search for feature conjunctions in adults with either good or poor phonological decoding skills. Although Buchholz and McKone (2004) showed reduced accuracy as a function of set size in adults with poor phonological skills, no previous study has investigated search times among this population. Furthermore, whereas,

abnormal search times have been shown for conjunction searches among dyslexic populations (Vidyasagar & Pammer, 1999), no previous study has examined the performance of dyslexics or poor phonological decoders on conjunction tasks involving the feature of motion. The detection of motion requires the involvement of the M-mediated Area MT of the PPC and prior research has implicated motion sensitivity deficits (Cornelissen et al., 1995), and the functioning of Area MT in the aetiology of dyslexia (Demb et al., 1998a; Demb et al., 1997, 1998b; Eden et al., 1996). Findings of parallel search functions for conjunctions involving the features of form and motion (McLeod, Driver, & Crisp, 1988) suggest that a “movement filter” acts to segregate the visual array into moving and stationary items, resulting in a simple parallel search of moving items. Bilateral lesions to the analogue of Area MT in humans produces deficits in restricting visual attention to moving items in visual search arrays, suggesting Area MT is a possible locus for such a movement filter (McLeod, Heywood, Driver, & Zihl, 1989).

The two-feature conjunction tasks used in the present study are similar to those used by Vidyasagar and Pammer (1999), however, smaller distractor set sizes were used, and colour/motion, and form/motion conjunction tasks were investigated in addition to the colour/form task. Based on previous findings in dyslexic populations (Vidyasagar & Pammer, 1999), poor phonological decoders are expected to show a greater increase in search times as a function of distractor set size when compared to good phonological decoders for the colour/form conjunction task. Considering the findings of decreased motion sensitivity among dyslexic populations (e.g., Cornelissen et al., 1995), and the relationship observed between motion thresholds and conjunction search performance (Illes et al., 2000), group differences are expected to be greatest when motion is included as a feature to be detected in conjunction search. Further, this difference is expected to be greater for the form/motion conjunction task in which reliance on Area MT is greatest, in comparison to the colour/motion task in which the additional feature of colour increases the saliency of potential targets.

The present study also aimed to investigate performance of good and poor phonological decoders on triple-feature conjunction tasks involving the search for moving or stationary targets. Search for targets defined by three features should be faster than search defined by two features due to the extra excitation received by items containing more than one of the attended feature (Wolfe et al., 1989). Search for a moving target among stationary distractors is easier than the reverse situation (Müller & Maxwell, 1994) suggesting that the natural bias of the movement filter to pass moving items rather than filter them out. However, the ability to search for stationary targets among moving distractors improves with practice, suggesting that the efficiency of search can be increased through suppressing the default “positive tagging” of motion signals to “negative tagging” (Müller & von Muhlenen, 1999). If there are greater differences observed between the groups on either the moving or stationary detection task, this will indicate differences in the ability to either positively or negatively tag motion signals respectively.

Method

Participants

The study was approved the University of Tasmania Human Research Ethics Committee. Forty first year psychology students at the University of Tasmania participated in this experiment as part of their course requirements. One male in each group was left handed and the remaining participants were right handed. All participants had normal or corrected to normal vision. Exclusion criteria included a history of drug, alcohol, or tobacco abuse, psychiatric or neurological disorder, head trauma, seizure, and those currently receiving medication. Participants were allocated to either a poor phonological decoder group (10F, 10M) or a good phonological decoder control group (10F, 10M) on the basis of scores on the Martin and Pratt Non Word reading test (Martin & Pratt, 2001). The scores of good phonological decoders ranged from 47-54 (out of a possible score of 54) and the scores of poor phonological decoders ranged

from 23–43. Norms for the Nonword reading test are available for samples up to 17 years of age (Martin & Pratt, 2001). The mean score of good decoder group was in the 81st percentile (> 17 years reading age equivalent) and the mean score of poor decoders was in the 18% percentile (10-11 years reading age equivalent) of this norming group.

Several reading and neuropsychological measures were administered in a screening session of approximately one hour on a day prior to the experimental session. Ravens Advanced Progressive Matrices (APM) was administered as a measure of non-verbal general intelligence (Raven, Court, & Raven, 1994). Other reading measures included the Word Identification and Comprehension subtests from the Woodcock-Johnson (WJ) Reading Mastery Tests (Woodcock, 1987), The National Adult Reading Test (NART: Nelson & Willison, 1991), an irregular word reading test (see Appendix A), and Reading Accuracy and Reading Rate measures from the Neale Analysis of Reading Ability. Other measures included the Digit Span, Vocabulary, Symbol Coding and Symbol Copy sub-tests from the Weschler Adult Intelligence Scale (WAIS-III: Wechsler, 1997).

Table 1 shows mean raw scores for each group on the reading and cognitive measures. No between group differences were found in terms of age, nonverbal intelligence (APM) and speed of processing measures such as symbol coding and symbol copy ($ps>.05$). However, poor phonological decoders showed significantly lower mean raw scores on measures of single word reading (nonwords, regular, and irregular words), passage comprehension, vocabulary, and reading accuracy and rate.

Table 1

Mean age and raw scores on reading and cognitive measures for good and poor phonological decoders in Experiment 1.

	Good Decoders		Poor Decoders		
	(n=20)		(n=20)		
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	Sig.
Age	19.83	2.39	19.46	2.50	<i>ns</i>
APM /36	24.95	5.54	22.50	3.72	<i>ns</i>
MP Nonword reading /54	50.30	1.62	35.35	5.93	***
WJ Word Identification /106	97.15	2.41	87.95	4.37	***
Irregular word reading /87	77.45	5.09	65.30	7.10	***
NART /50	31.10	1.08	19.40	4.33	***
DS Forwards [#] /16	11.40	1.93	9.90	1.74	*
DS Backwards [#] /14	7.95	2.04	6.45	1.76	*
WJ Comprehension [†] /68	59.75	3.18	56.80	4.62	*
WAIS Vocabulary [†] /66	50.25	6.23	43.95	6.86	**
WAIS Symbol Coding [†] /60	79.90	8.29	75.35	6.69	<i>ns</i>
WAIS Symbol Copy [†] /133	122.45	15.10	118.7	14.47	<i>ns</i>
Neale Reading Accuracy [†] (%)	98.00	2.87	89.60	6.16	***
Neale Reading Rate [†] (words/min)	142.05	15.85	115.1	18.28	***

Note: * $p < .05$, ** $p < .01$, *** $p < .001$, [#] Missing data was substituted with the mean for the group for one good decoder, [†] Missing data was substituted with the mean for the group for three good decoders.

Stimuli/Apparatus

Stimuli were presented on a BARCO monitor driven by a VSG card and interfaced with an IBM computer. Tasks were custom programmed in Visual Basic for Windows. A two-button response pad was used to initiate blocks of trials and to make responses. Viewing distance was kept constant at a distance of 60cm using a chin rest. The laboratory was dimly lit. Colour displays were isoluminant and presented on a black background under mesopic lighting conditions. Individual stimulus elements were 0.5cm in size ($\sim 0.48^\circ$ of visual angle) and all items were presented at randomly chosen locations within an invisible grid consisting of 150 locations and subtending a region of approximately $13.7^\circ \times 9.2^\circ$ of visual angle. All stimuli were plotted with a minimum of 0.5cm ($\sim 0.48^\circ$) between them. All moving stimuli were displaced 0.5cm ($\sim 0.48^\circ$) from left to right, spending 200ms at each position and as such oscillating at approximately 2.5Hz (.025m/s).

Two blocks of 48 trials were administered for each of five tasks. Stimuli in one block of trials were red and green and in the other block they were yellow and purple. For 16 trials in each block, distractor set size was 6, 12, or 18 items. Target stimuli were positioned randomly within the grid on 50% of trials and were absent on the remainder of trials. The five tasks required the search for conjunctions of colour, form, or motion features as follows: Colour/form (CF): search for a green triangle among green circles and red triangles; Colour/Motion (CM): search for a moving green triangle among stationary green triangles and moving red triangles; Form/Motion (FM): search for a moving green triangle among stationary green triangles and moving green circles; Colour/ Form/ Motion (CFM): search for a moving green triangle among stationary green triangles and moving red circles; Colour/Form/ Motion stationary target (CFMS): search for a stationary green triangle among moving green triangles and stationary red circles.

Procedure

In an experimental session lasting approximately thirty minutes, participants were required to search for a target based on the conjunction of two or three features among an array of distractor items and to respond 'yes' or 'no' to the presence of the target using a two key response pad. Prior to the commencement of the experimental trials, participants completed two blocks of 10 practice trials, requiring a simple search for a grey triangle among grey circles presented on a black background. The five experimental tasks were administered in counterbalanced order. Prior to commencement of each block of trials, a brief verbal description was given, and a description of the target stimuli was presented on the screen. Participants were instructed to focus their eyes on the fixation cross between trials and to respond as quickly and accurately as possible. Response times were measured from stimulus onset and each trial remained on the screen until a response was made. Response times less than 200ms or greater than 5000ms were excluded as being either anticipatory or too long respectively.

Design and Data Analysis

The experiment followed a 2 [Group: good decoders, poor decoders] x 3 (Set size: 6, 12, 18) x 2 (Target: present, absent) x 5 (Task: CF, CM, FM, CFM, CFMS) mixed design, with mean search times for correct trials and accuracy as the dependent measures. Analyses conducted with Sex as an additional between subjects factor are not reported as there were no significant effects involving sex. Data were analysed using repeated measures ANOVAs with Greenhouse-Geisser corrections to control for violations of the assumption of sphericity. Significant interactions were further analysed using univariate ANOVAs with Bonferroni adjustments. Planned one-way between subjects ANOVAs were conducted for each task when the target was either present or absent. For target present trials, planned analyses were conducted to examine the Set size x Group interaction for each task. To investigate any differences in the positive or negative

tagging of motion signals among the two groups, a planned four-way ANOVA was also conducted including only the two three-feature conjunction tasks (CFM, CFMS).

Results

Mean Search Times

For correct trials, overall search times were significantly shorter for target present ($M=1013.18$, $SEM=18.68$) in comparison to target absent ($M=1174.9$, $SEM=29.54$) trials, $F(1,38)=107.53$, $MSE=72980.9$, $p<.001$, and increased significantly across Set size, $F(2,76)=204.19$, $MSE=82069.3$, $p<.001$. Figure 1 shows the significant main effect of Task, $F(4,152)=54.31$, $MSE=144732.9$, $p<.001$. Mean search times were shortest for the CF task followed by CFMS, CFM, CM and FM tasks. All differences between tasks were significant ($p<.01$) except for the difference between the CM and CFM tasks ($p>.05$).

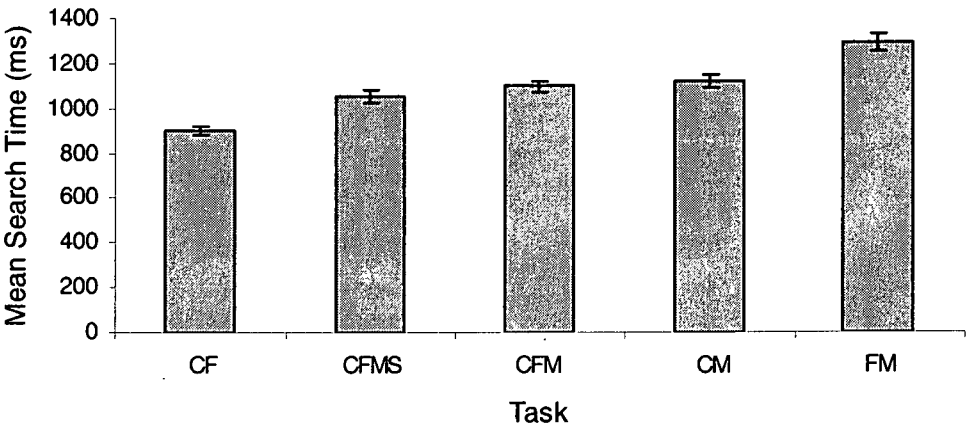


Figure 1. Mean search times for conjunction search tasks (CF=colour/form; CFMS=colour/form/motion (stationary); CFM=colour/form/motion; CM=colour/motion; FM=form/motion).

Overall search times were significantly slower for poor decoders ($M=1150.7$, $SEM=33.2$) in comparison to good decoders ($M=1037.4$, $SEM=33.2$), $F(1,38)=5.84$, $MSE=659867.2$, $p<.05$. This was qualified by a significant Response x Group interaction, $F(1,38)=6.60$, $MSE=72980.9$, $p<.05$, such that the effect of Group was significant for target absent trials, $F(1,38)=6.74$, $MSE=523583.7$, $p<.025$ (Bonferroni corrected), and only approached significance for target present trials, $F(1,38)=3.84$, $MSE=209264.3$, $p=.057$. The Task x Group and Set size x Group interactions were non significant ($ps>.05$), and there was a trend for a Response x Task x Group interaction, $F(4,152)=2.19$, $MSE=33854.2$, $p=.099$ (see Figure 2). Group differences were greater when the target was absent, particularly for the FM and CFM tasks.

Planned comparisons were conducted to examine overall group differences for each task when the target was present or absent. For target present trials the main effect of Group was significant for the FM task, $F(1,38)=5.52$, $MSE=71587.9$, $p<.05$, and approached significance for the CFM, $F(1,38)=3.56$, $MSE=63130.7$, $p=.067$, and the CFMS, $F(1,38)=3.39$, $MSE=58887.5$, $p=.074$ tasks. For target absent trials, the main effect of Group was significant for the FM task, $F(1,38)=6.89$, $MSE=365848.7$, $p<.05$, and the CFM task, $F(1,38)=7.45$, $MSE=110427$, $p<.05$, approached significance for the CM task, $F(1,38)=3.39$, $MSE=149542$, $p=.073$, and was non significant for the CFMS, $F(1,38)=2.36$, $MSE=176159.4$, $p>.05$, and the CF, $F(1,38)=1.76$, $MSE=69308.4$, $p>.05$ tasks.

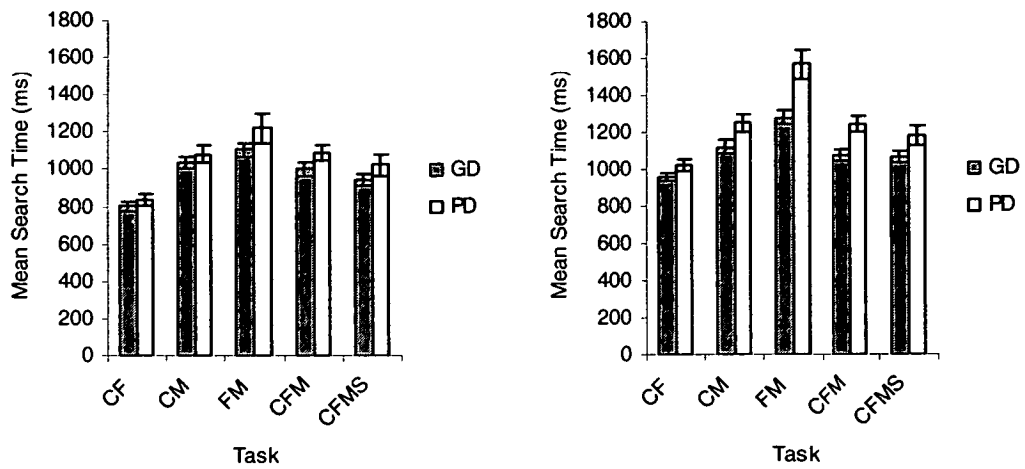


Figure 2. Mean search times for good decoders (GD) and poor decoders (PD) as a function of task for target present (left) and target absent (right) trials (CF=colour/form; CFMS=colour/form/motion (stationary); CFM=colour/form/motion; CM=colour/motion; FM=form/motion).

When target present trials were analysed separately there was a trend for an overall Set size x Group interaction, $F(2,76)=2.98$, $MSE=13219.0$, $p=.065$. Planned comparisons were performed to examine the Set size x Group interaction for each task. Figure 3 (left) shows a significant Set size x Group interaction for the CF task, $F(2,76)=4.11$, $MSE=3329.503$, $p<.05$. The effect of Group tended to be significant for a set size of 12 items, $F(1,38)=4.08$, $MSE=14277.8$, $p=.05$, and not for set sizes of six or 18 items ($ps>.05$). Figure 3 (right) also shows a trend for Set size x Group interaction for the FM task, $F(2,76)=3.01$, $MSE=9396.3$, $p=.056$. The effect of Group approached significance for 12, $F(1,38)=5.36$, $MSE=34322.5$, $p=.026$ ($p>.05$, Bonferroni corrected), and 18 items, $F(1,38)=5.82$, $MSE=40687.7$, $p=.021$ ($p>.05$, Bonferroni corrected), but was non-significant for six items ($p>.05$).

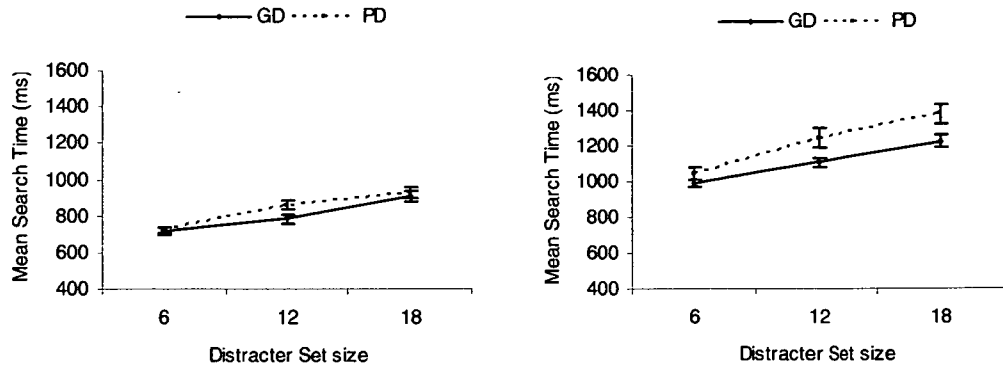


Figure 3. Mean search times for good decoders (GD) and poor decoders (PD) as a function of distracter set size for the CF (left) and FM (right) tasks.

A planned ANOVA including only the CFM and CFMS tasks was conducted to investigate any Group differences in the positive or negative tagging of motion signals. There was a significant main effect of Group, $F(1,38)=4.89$, $MSE=313005.2$, $p<.05$, indicating significantly greater search times for poor decoders ($M=1132.6$, $SEM=36.1$) in comparison to good decoders ($M=1019.8$, $SEM=36.1$), but this effect did not interact significantly with any other factor ($ps>.05$).

Accuracy

The main effect of Group was non-significant ($p>.05$). Mean accuracy was greater for target absent ($M=14.99$, $SEM=.092$) in comparison to target present ($M=15.74$, $SEM=.027$) trials, $F(1,38)=74.96$, $MSE=2.41$, $p<.001$. Accuracy was found to decrease as a function of Set size, $F(2,76)=21.51$, $MSE=1.25$, $p<.001$, however, this was modified by a significant Response x Distractor interaction, $F(2,76)=36.25$, $MSE=0.992$, $p<.001$. The main effect of Set size was significant for target present trials, $F(2,76)=19.84$, $MSE=1.99$, $p<.001$, such that accuracy was significantly lower for set sizes of 12 and 18 in comparison to six items ($p<.05$). The effect of

Set size was non-significant for target absent trials ($p > .05$). There were no significant two-way or three-way interactions involving Task or Group ($ps > .05$).

Discussion

This study investigated the ability of good and poor adult phonological decoders to search visually for targets defined by the conjunctions of the features of colour, form, and motion. Consistent with previous findings, search times were faster when targets were present compared to when they were absent (Treisman & Gelade, 1980). Search times increased significantly across distractor size set for all tasks. According to feature integration theory, this indicates a serial self-terminating search rather than parallel processing across the entire visual scene (Treisman, 1988; Treisman & Gelade, 1980). The fastest search times were found for the colour/form conjunction task overall followed by triple-feature conjunction tasks for stationary and moving targets respectively. The two-feature conjunction search for form among a moving array of distractor items of the same colour proved to be the most difficult task. For tasks involving motion, search times were faster for the CFM in comparison to the CM and FM tasks. This is consistent with previous research showing faster search times for triple relative to two feature conjunction tasks and reflects the additional benefit in the guidance of spatial attention by parallel processing of individual features (Wolfe et al., 1989).

Poor phonological decoders showed significantly longer search times in comparison to good phonological decoders and this could not be attributed to a speed accuracy trade-off as there were no significant effects involving group in the accuracy data. It is also unlikely that group differences can be attributed to longer speed of processing times among poor decoders as there were no group differences on speed of processing measures such as symbol search and symbol copy. Findings of differences in search times for conjunction tasks among adult poor phonological decoders is consistent with other studies showing longer conjunction search times among both children (Vidyasagar & Pammer, 1999) and adult developmental dyslexics (Illes et

al., 2000) relative to controls. According to feature integration theory, the search for feature conjunctions requires the guidance of focal attention in order to conjoin different features occurring at the same location. Thus, differences between good and poor decoders in conjunction search times may reflect differences in control of spatial attention.

Also consistent with previous research (e.g., Buchholz & McKone, 2004; Illes et al., 2000; Vidyasagar & Pammer, 1999), differences between the two groups increased as a function of distractor set size, particularly for the form/motion task. Illes et al. (2000) reported several significant Set size x Group interactions among adult dyslexics using a similar set size (1, 2, 4, 8 or 16 items) to the present study. Furthermore, in a study in which accuracy rather than search times were emphasised, adults with poor phonological processing skills showed a greater decrease in accuracy across distractor set size (5, 7, 11 items) in comparison to control subjects (Buchholz & McKone, 2004). These findings indicate that poor decoders have difficulty with selectively processing items while excluding unattended items. According to modifications of Feature Integration Theory, both excitatory and inhibitory influences are used to guide spatial attention during conjunction searches search (Treisman, 1988; Treisman & Sato, 1990; Wolfe, 1994, 1998; see Wolfe et al., 1989). However, it is not possible to determine from the present study whether group differences occurred due to less excitation of relevant locations and/or less inhibition of irrelevant locations and therefore greater distraction. Furthermore, according to Attentional Engagement Theory (e.g., Duncan, 1996; Duncan & Humphreys, 1989) top-down attentional control or perceptual grouping mechanisms could also have contributed to the findings.

Findings of impairment on conjunction search tasks among dyslexics and poor phonological decoders are consistent with the possibility of impairment within the M-mediated PPC (Stein & Walsh, 1997). It has been argued that attentional feedback from the PPC to the striate cortex acts to gate input to the ventral visual processing stream, providing a spotlighting function for relevant locations and aiding the serial search for conjunctions (Vidyasagar, 1999,

2001, 2004). This spotlighting function may be particularly important for the serial allocation of attention to letters and words of text during reading. The largest group difference was observed for the form/motion conjunction task which is consistent with research showing reduced sensitivity to motion among dyslexic populations (Cornelissen & Hansen, 1998; Cornelissen et al., 1995). Further, Illes et al. (2000) found a relationship between elevated motion thresholds and performance on visual search tasks involving conjunctions among dyslexic adults. The form/motion task used in the present study is more likely to involve the M-mediated parietal visual processing stream in comparison to the other tasks, as it requires sensitivity to motion as well as the serial allocation of attention. This is also the only task in which the parvocellular mediated ventral stream did not contribute to processing by coding the salient feature of colour.

Another aim of the study was to investigate differences in the positive and negative tagging of motion signals using triple conjunction tasks in which the target was in either the moving or stationary set respectively. However, whereas previous research has shown that search for a moving target among stationary distractors is easier than search for a stationary target among moving distractors (Müller & Maxwell, 1994), this finding was not replicated in the present study. It is possible that the movement speed used in the present study was too slow to examine the quick parallel integration of motion signals effectively. Thus although the present findings indicate no differential effect of positive or negative tagging of motion signals among good and poor phonological decoders, further research is required to investigate this further.

Overall group differences in search times were greatest for target absent trials, and particularly for the form/motion task. Serial self-terminating visual search models (Treisman & Gelade, 1980) predict that deciding that the target is not present will take longer because it requires search of the entire array rather than termination of the search upon identification of the target. According to parallel competitive models (Duncan & Humphreys, 1989), facilitation of the target builds up more slowly with a greater number of elements, thus whereas the presence of a target relies on the increase in signal to noise ratio of the target, deciding on the absence of a

target requires the ability to discard signals as noise rather than task relevant signals. Poor decoders may have performed more poorly on target absent trials due to a number of factors including a slower and/or less efficient self terminating search, lower signal to noise ratio, or less inhibition of irrelevant distractors.

Experiment 1 has shown that poor phonological decoders show longer search times for conjunction searches relative to good decoders, particularly for conjunction searches defined by the features of form and motion. These findings are consistent with results from visual search studies in dyslexia, and suggest differences in the functioning of spatial attention mechanisms that guide visual search. From the present research it is not possible to determine the specific attentional mechanisms that differ between the two groups. For example, the findings could be attributable to differential excitation of relevant target locations (Wolfe et al., 1989), inhibition of irrelevant locations (Treisman & Sato, 1990), or a combination of both. It is also possible that other attentional phenomena such as perceptual grouping, the size of the attentional focus, or attentional dwell or shift times could account for the present results. The following experiments aim to examine attentional processes among good and poor phonological decoders in more detail by examining specific mechanisms involved in spatial attention, and their relationship to components of the ERP waveform (e.g., N1, P1, N2) that index early attentional and perceptual processes.

CHAPTER 7 - EXPERIMENT 2: ERP CORRELATES OF FLANKER AND CUE-SIZE EFFECTS AMONG GOOD AND POOR ADULT PHONOLOGICAL DECODERS.

Attention can be allocated evenly across the visual field such that visual stimuli are processed in parallel, or can be focused to facilitate processing at one location and inhibit processing at other locations (e.g., Jonides, 1983). Selective attention to individual words and letters and the suppression of those that are not the current focus of attention may be particularly important for the process of phonological decoding during reading (LaBerge & Brown, 1989). Thus, the present study aimed to investigate both behavioural and electrophysiological correlates of attentional focussing and inhibition in good and poor adult phonological decoders.

The facilitation of attention at particular spatial locations is often likened to a 'spotlight' of a specific size (James, 1950; Posner, 1980; Posner et al., 1980b). However, processing is facilitated when targets are preceded by small in comparison to large cues. This 'cue-size effect' is consistent with a 'zoom lens' model of attention (Eriksen & St James, 1986) and suggests a processing speed advantage for a narrow versus diffuse attentional focus (Castiello & Umiltà, 1990; Castiello & Umiltà, 1992). Separate mechanisms have been proposed for moving or shifting spatial attention (orienting) (Posner, 1980; Posner et al., 1980b) and adjusting the size of attentional focus (focusing) (Benso et al., 1998). The cue-size effect increases as a function of SOA from 40-50ms with maximal facilitation observed at 500ms (Castiello & Umiltà, 1990). Thus, it has been argued that attentional focussing consists of an automatic process that is triggered by the onset of a stimulus, and a later stage during which the size of the attentional focus is maintained voluntarily, followed by a more diffuse mode of attention (see Benso et al., 1998; Turatto et al., 2000). The N1 component of the ERP waveform is modulated by spatial attention during covert orienting such that greater amplitude is observed for valid relative to invalid trials (for reviews see Eimer, 1998; Mangun, 1995). The N1 attention effect is thought to reflect the gradient of visual attention (Mangun & Hillyard, 1991) or the demands of visual discrimination within the focus of attention (Vogel & Luck, 2000). Consistent with this proposal

attentional modulation of the N1 component has been found as a function of cue-size manipulations such that amplitude is greater for small relative to large cues (Luo, Greenwood, & Parasuraman, 2001).

Studies that have investigated attentional focussing among developmental dyslexics have revealed conflicting findings. In a study using a simple dot detection task presented at fixation, dyslexic children ($n=10$) showed significant cue size effects at short (99ms) but not at long (504ms) SOAs, whereas the cue-size effect was significant for both SOAs for normally reading children ($n=10$) and adults ($n=10$) (Facoetti et al., 2000b). It was suggested that dyslexics were not able to maintain or sustain active focussing over time and had shifted to a more distributed and less efficient visual processing mode. However, Facoetti et al. (2003a) investigated cue-size effects in dyslexic ($n=10$) and normally reading ($n=13$) children using a variable SOA of 100ms or 500ms. Participants were required to respond to the direction of a central arrow preceded by a small or large cue. Whereas control children showed cue-size effects at both SOAs, dyslexic children showed a cue-size effect at the long (500ms) but not the short (100ms) SOA, suggesting a deficit in the automatic focussing of spatial attention (Facoetti et al., 2003a). The authors suggested that the different findings of these two studies could be due to task difficulty, perceptual load, or the allocation of processing resources (Facoetti et al., 2003a).

As well as perceptual enhancement of target locations, inhibitory mechanisms act to suppress information in unattended areas of the visual field (Cepeda, Cave, Bichot, & Kim, 1998). The ability to suppress irrelevant information that is not the current focus of attention can be investigated using 'flanker' paradigms in which participants respond to a central target stimulus that is flanked by either response compatible or incompatible distractor letters (Eriksen & Eriksen, 1974). The 'flanker effect' refers to the increase in RT observed for targets surrounded by incompatible distractors relative to baseline and the decrease in RT for compatible flankers relative to baseline. Eriksen and Eriksen (1974) attributed the flanker effect to interference at the response selection stage, postulating that the effect occurred due to the time

taken to inhibit an incompatible response. Electrophysiological studies have identified a fronto-central N200 component that is elicited in response to flanker stimuli that are response incompatible (Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988; Heil et al., 2000; Kopp et al., 1996b). This frontal N200 is thought to reflect inhibition of automatically primed but irrelevant responses by the frontal executive attentional network (Heil et al., 2000; Kopp et al., 1996b), but may also play a role in directing attention towards task relevant events (Weissman et al., 2005).

Other research suggests that early selection mechanisms play a role in flanker inhibition and a relationship has been observed between flanker effects and spatial attention. For example, a reduction in the flanker effect is found when spatial attention is narrowly focussed on the target (LaBerge et al., 1991; Yantis & Johnston, 1990) suggesting that flanker effects occur due to attentional leakage to the flanker location when early selection is not completely or efficiently focussed (Yantis & Johnston, 1990). Similarly, an increase in the flanker effect has been found when targets appear following invalid relative to valid or no cue conditions. These findings suggest that the ability to ignore flankers is reduced when a shift in spatial attention is required, and indicates an interaction between orienting and inhibitory executive function (Callejas, Lupianez, & Tudela, 2004). Facoetti and Molteni (2000) investigated whether attentional focussing acts as an inhibitory mechanism for suppressing distractors. The size of attentional focus was manipulated with small or large cues presented either simultaneously or 500ms prior to target onset. It was hypothesised that the effect of compatibility would be greater when flankers fell inside the attended area (large cue-size) compared to when flankers fell outside (small cue-size), particularly for the 500ms SOA condition during which attention is optimally focussed. A reduction in the flanker effect was found for small cues in the 500ms SOA condition, suggesting that irrelevant distractor locations are inhibited when spatial attention is optimally focussed.

Several lines of research suggest that reading disability is associated with difficulty inhibiting distracting visual information (Bednarek et al., 2004; Brosnan et al., 2002; Facoetti &

Turatto, 2000; Klein & D'Entremont, 1999). Using a number identification task, Klein and D'Entremont (1999) found that adult psychology students who were poor readers did not show the same decrease in the size of the flanker effect as a function of flanker eccentricity as good readers. Bednarek et al. (2004) investigated the processes of alerting, orienting, and inhibition (or resolution of conflict) among Spanish dyslexics and normally reading children using a cueing task in which targets were surrounded by compatible and incompatible flanking stimuli. Dyslexics were found to be impaired relative to controls on accuracy and RT measures when targets were flanked by incompatible flankers suggesting a deficit in executive control rather than an orienting or alerting deficit. It was argued that the origin of this effect could be either executive function mediated by the prefrontal cortex, attentional processes mediated by the PPC, or the magnocellular visual processing stream which has been linked to the identification of flanker stimuli (Omtzigt & Hendriks, 2004; Omtzigt et al., 2002).

The aim of the present study was to examine both attentional focussing and inhibition of unattended visual stimuli among good and poor adult phonological decoders using a modification of the flanker task. A go/nogo paradigm was used rather than a two-choice response task in order to reduce the influence of response competition and increase the influence of perceptual and attentional processes. To investigate focusing of spatial attention, target stimuli were preceded by either large or small cues that were presented either simultaneously or 500ms prior to the target (cued). Cue-size effects are expected to be greater for the cued in comparison to the simultaneous condition (Benso et al., 1998; Castiello & Umiltà, 1990) and flanker effects are expected to be greater for large in comparison to small cues (Callejas et al., 2004; Facoetti & Molteni, 2000). The greatest reduction in the flanker effect is expected for the 500ms SOA condition when attention is optimally focussed by a small cue allowing for the inhibition of the irrelevant flankers (Facoetti & Molteni, 2000).

A further aim of the present experiment was to examine electrophysiological correlates of attentional processing within both the posterior attentional network involved in focussing

spatial attention and the frontal executive control network involved in the inhibition of task irrelevant information and responses. Posterior N1 amplitude is expected to be greater when targets are presented with small relative to large cues (e.g., Luo et al., 2001; Mangun, 1995), particularly for the 500ms SOA condition. Further, if the focussing of spatial attention plays a role in the inhibition of irrelevant flankers N1 amplitude is expected to be greater in the presence of incompatible flanker stimuli preceded by large cues. Considering the findings that frontal N200 amplitude represents the inhibition of response irrelevant processing (e.g., Heil et al., 2000; Kopp et al., 1996b), frontal N200 amplitude is expected to be greater when targets are surrounded by incompatible flankers and reduced when flanker stimuli are response compatible.

A reduction in the cue-size effect for poor phonological decoders would be consistent with impaired attentional focussing and may be accompanied by a reduction in the N1 attention effect. If poor phonological decoders have difficulty ignoring distracting visual information they are expected to show a greater reaction times to target stimuli preceded by large cues and flanked by incompatible stimuli. A reduction in the ability to focus on the target under these conditions may be accompanied by a reduction in posterior N1 amplitude implicating the posterior attentional network. In contrast, a reduction in the N200 flanker effect would implicate a deficit of inhibitory control within the frontal attentional network.

Method

Participants

The study was approved the University of Tasmania Human Research Ethics Committee. Thirty two first year psychology students at the University of Tasmania participated in the experiment as part of their course requirement and all gave written informed consent prior to participation. One male and one female from each group were left handed, the remaining participants were right handed as measured by the Edinburgh handedness inventory (Oldfield, 1971). All participants had normal or corrected to normal vision and exclusion criteria included

a history of drug, alcohol, or tobacco abuse, psychiatric or neurological disorder, head trauma, seizure, and those currently receiving medication. Participants were selected from a sample of over three hundred students on the basis of Martin and Pratt Nonword Reading Test scores (Martin & Pratt, 2001). A brief in-class screening was conducted using Form B, followed by a more detailed reading assessment using Form A of the test. The sample consisted of 17 good decoders (10 female, 7 male) and 14 poor decoders (8 female, 6 male). The scores of good phonological decoders ranged from 49-54 (out of a possible score of 54) and the scores of poor phonological decoders ranged from 19-43. Norms for the Nonword reading test are available for samples up to 17 years of age (Martin & Pratt, 2001). The mean score of good decoder group was in the 81st percentile (> 17 years reading age equivalent) and the mean score of poor decoders was in the 18th percentile (10-11 years reading age equivalent) of this norming group. One female poor decoder was excluded due to overall accuracy below 70% on the experimental tasks.

Several reading and neuropsychological measures were administered in a screening session of approximately one hour on a day prior to the experimental session. Ravens Advanced Progressive Matrices (APM) was administered as a measure of non-verbal general intelligence (Raven et al., 1994). Other reading measures included the Word Identification and Comprehension subtests from the Woodcock-Johnson (WJ) Reading Mastery Tests (Woodcock, 1987), The National Adult Reading Test (NART: Nelson & Willison, 1991), an irregular word reading test (see Appendix A), and Reading Accuracy and Reading Rate measures from the Neale Analysis of Reading Ability. Other measures included the Digit Span, Vocabulary, Symbol Coding and Symbol Copy sub-tests from the Weschler Adult Intelligence Scale (WAIS-III: Wechsler, 1997).

Table 2 shows mean age, APM raw score, and raw scores on other reading and cognitive measures for each group. No between group differences were found in terms of age, general nonverbal intelligence (APM), or digit span backwards. Good decoders had significantly higher

raw scores in comparison to poor decoders on reading measures including: non-word reading, WJ word identification, irregular word reading, the NART, digit span forwards, WJ comprehension, Vocabulary, symbol coding, and Neale reading rate and accuracy. Good decoders also tended to have higher symbol copy scores in comparison to poor decoders ($p=.055$). A ceiling effect was observed for non-word reading scores, as the upper range for inclusion into the study was much smaller than the lower range to ensure large differences between groups.

Stimuli and Apparatus

Stimuli were presented on an IBM computer and tasks were programmed using the NeuroScan STIM program. Each trial began with a 300ms presentation of a central fixation cross, followed immediately by presentation of a circular cue (small or large) which remained on the screen throughout the 200ms presentation of a target stimulus composed of three lowercase letters (b or d). Small cues created a circle around the middle letter of the target stimulus and large cues created a circle around all three letters. The middle letter of the target stimulus was either a 'b' or 'd' on 50% of trials and was randomly surrounded by either compatible (same letter) or incompatible (other letter) on an equal proportion of trials. The cue and the target stimulus appeared either simultaneously (0ms SOA) or with a 500ms cue to target SOA. Stimuli were presented white on a black background and each letter subtended 1 x 1 degrees of visual angle at a viewing distance of 70cm, with 1° spacing between each letter. The inter-trial interval was 1000ms. There were four blocks of experimental trials, two for each SOA condition, and one block for each go/nogo stimulus (b or d).

Table 2

Mean age and raw scores on reading and cognitive measures for good and poor phonological decoders in Experiment 2.

	Good Decoders		Poor Decoders		Sig.
	(n=17)		(n=14)		
	M	SD	M	SD	
Age	21.64	3.85	19.86	2.40	ns
APM /36	23.71	5.18	21.50	3.84	ns
MP Nonword Reading /54	50.65	1.50	35.14	6.92	***
WJ Word Identification /106	98.41	2.62	88.00	5.14	***
Irregular word reading /87	78.47	8.89	60.71	7.82	***
NART [#] /50	33.82	4.20	18.21	4.23	***
Digit Span Forwards [#] /16	11.71	1.72	9.29	2.37	**
Digit Span Backwards [#] /14	8.00	1.58	7.29	2.30	ns
WJ Comprehension /68	61.1	2.5	55.9	4.6	***
WAIS Vocabulary [†] /66	55.3	5.3	42.9	7.3	***
WAIS Symbol Coding [†] /60	81.2	8.5	74.8	8.4	*
WAIS Symbol Copy [†] /33	125.6	10.3	115.8	16.7	p=.055
Neale Reading Accuracy [†] (%)	98.2	2.1	88.1	5.6	***
Neale Reading Rate [†] (words/min)	147.6	16.3	110.4	11.6	***

Note: * $p < .05$, ** $p < .01$, *** $p < .001$, [#] Missing data was substituted with the mean for the group for two poor decoders and two good decoders, [†] Missing data was substituted with the mean for the group for five good decoders and three poor decoders.

Electrophysiological Recording

EEG activity was recorded with a NeuroScan system, consisting of a 32-channel Synamps, SCAN 4.1 software, and Quik-cap with Ag/AgCl electrodes interfaced with a NeuroScan STIM 3.1 computer. EEG was recorded from 32 sites, according to the international 10-20 system, and all electrodes were referenced to the mastoids. Horizontal electro-oculographic (EOG) activity was recorded bipolarly from electrodes at the outer canthi of both eyes, and vertical EOG was recorded from electrodes above and below the left eye. Electrode impedance was kept below 5 k Ω . EEG activity was amplified with a band pass of 0.15-100 Hz and sampled continuously at a rate of 1000 Hz. Continuous EEG files were merged with behavioural files and subjected to a zero phase-shift band-pass filter (0.15-30Hz, 24dB rolloff). Ocular artifact reduction was performed by regression and artifact averaging (Semlitsch, Anderer, Schuster, & Presslich, 1986). Data files were epoched offline for a 1000ms epoch commencing 100ms before stimulus onset and baseline corrected. High and low voltage cut-offs for artifact rejection were set at 100 μ V and -100 μ V respectively. Correct responses were averaged for each stimulus type and then band-pass filtered (0.15-30 Hz). Posterior N1 and P1 and anterior N2 components were determined from grand averaged means as the maximum voltage within the following time frames after target stimulus onset: Posterior N1: 125-185-200ms; Anterior N2: 210-300ms.

Procedure

Following set-up for EEG recording, participants were seated in front of a computer monitor, at a viewing distance of 70cm. Four go/nogo letter discrimination tasks were completed in counterbalanced order. Participants were required to respond when the middle letter of the target stimulus was the Go stimulus (b or d) and to withhold responses for the nogo stimulus (b or d) using a response pad. Participants were instructed to respond as quickly and as accurately as possible to all 'go' stimuli irrespective of cue size or flanker compatibility and to avoid

blinking or any other overt movements. Participants took short breaks between tasks to prevent fatigue. The experimental session lasted approximately two hours (including set up for electrophysiological recording) with most participants completing several different experiments within the session.

Design and Data Analysis

Mean RT, accuracy, and ERP waveforms were averaged across each letter type (b, d). The effects of experimental manipulations on mean RT were investigated using a 2[Group: good decoder, poor decoder] x 2[Sex: male, female] x 2(SOA: 0ms, 500ms) x 2(Cue size: small, large) x 2(Flanker: compatible, incompatible) repeated measures ANOVA with two between subjects factors. An additional factor of 2(Response: go, nogo) was included for analyses of mean accuracy. The effects of experimental manipulations on posterior N1 amplitude and latency were analysed using a 2[Group: good decoder, poor decoder] x 2[Sex: male, female] x 2(SOA: 0ms, 500ms) x 2(Cue size: small, large) x 2(Flanker: compatible, incompatible) x 2(Hemisphere: left, right) x 3(Sagittal site: occipital, parietal, temporal) repeated measures ANOVA with two between subjects factors. Anterior N2 amplitude and latency were analysed using a 2[Group: good decoder, poor decoder] x 2[Sex: male, female] x 2(SOA: 0ms, 500ms) x 2(Cue size: small, large) x 2(Flanker: compatible, incompatible) x 2(Site: F3, Fz, F4) repeated measures ANOVA. Greenhouse-Geisser corrections were applied where appropriate to control for violations of sphericity. Significant interactions were analysed using break-down ANOVAs for analysis of simple effects. Bonferroni adjusted p-values were used to maintain the family-wise Type 1 error rate and these are reported when the correction changes the significance of the analysis. Sex was included as a factor in all analyses but only effects in which Group interacted with Sex are reported.

Results

Mean Reaction Time

Table 3 shows mean RT (s) for good decoders and poor decoders for each experimental condition. Mean RT was significantly shorter for compatible ($M=0.368$, $SEM=.006$) than incompatible ($M=0.405$, $SEM=.007$) stimuli, $F(1,27)=86.16$, $MSE=.001$, $p<.001$, and for the 500ms SOA ($M=0.383$, $SEM=0.006$) than the 0ms SOA ($M=0.390$, $SEM=0.007$) condition, $F(1,27)=4.26$, $MSE=0.001$, $p<.05$. The main effect of Group was non-significant, $F(1,27)=2.09$, $MSE=0.01$, $p>.05$.

Table 3

Mean reaction time (s) for good and poor decoders for each experimental condition in Experiment 2.

SOA	Cue	Flanker	Good Decoders		Poor Decoders	
			(n=17)		(n=14)	
			<i>M</i>	<i>SEM</i>	<i>M</i>	<i>SEM</i>
0ms	Small	Compatible	.370	.012	.397	.013
		Incompatible	.404	.010	.409	.011
	Large	Compatible	.367	.010	.377	.011
		Incompatible	.400	.011	.419	.012
500ms	Small	Compatible	.360	.010	.379	.012
		Incompatible	.397	.011	.409	.013
	Large	Compatible	.346	.008	.370	.009
		Incompatible	.405	.010	.426	.011

There was a significant SOA x Flanker interaction, $F(1,29)=20.72$, $MSE= 0.0002$, $p<.001$. Mean RT was significantly greater for incompatible than compatible stimuli for both the 0ms SOA, $F(1,27)=41.16$, $MSE=0.0007$, $p<.001$, and the 500ms SOA condition, $F(1,27)=127.51$, $MSE=0.0005$, $p<.001$. However for compatible stimuli, mean RT was shorter for the 500ms in comparison to the 0ms SOA condition, $F(1,27)=14.58$, $MSE=0.0004$, $p<.01$.

The Cue x Flanker interaction was also significant, $F(1,27)=8.23$, $MSE=0.001$, $p<.01$. Mean RT was greater for incompatible in comparison to compatible stimuli for both small, $F(1,27)=17.14$, $MSE=0.001$, $p<.001$, and large cue types, $F(1,27)=307.52$, $MSE=0.0002$, $p<.001$. However, mean RT was significantly shorter for small in comparison to large cues for incompatible stimuli, $F(1,27)=6.11$, $MSE=0.0002$, $p<.05$, and tended to be greater for large in comparison to small cues for compatible stimuli, $F(1,27)=4.38$, $MSE=0.0008$, $p=.046$ ($p>.05$, Bonferroni corrected).

However, these two-way interactions were qualified by a significant SOA x Cue x Flanker x Group interaction, $F(1,29)=5.59$, $MSE=0.0001$, $p<.05$. In order to elucidate these effects separate analyses were conducted for each SOA condition and planned comparisons were conducted to investigate the effects of Cue and Flanker for each group and the main effect of Group for each condition. Figure 4 shows a significant Cue x Flanker x Group interaction for the 0ms SOA condition, $F(1,27)=4.97$, $MSE=0.0004$, $p<.05$. Good decoders showed shorter RT for compatible in comparison to incompatible flanker stimuli regardless of Cue, $F(1,15)=46.71$, $MSE=0.0004$, $p<.001$. In contrast, poor decoders showed a significant Cue x Flanker interaction, $F(1,12)=4.78$, $MSE=0.0006$, $p<.05$, such that RT was significantly greater for incompatible than compatible flankers when presented with a large cue, $F(1,12)=48.52$, $MSE=0.0002$, $p<.001$, but not when presented with a small cue ($p>.05$). Figure 4 shows that this effect is largely due to increased RT to compatible flankers presented with the small cue. The effect of Cue was non-significant for both groups, and there were no significant between group differences ($ps>.05$).

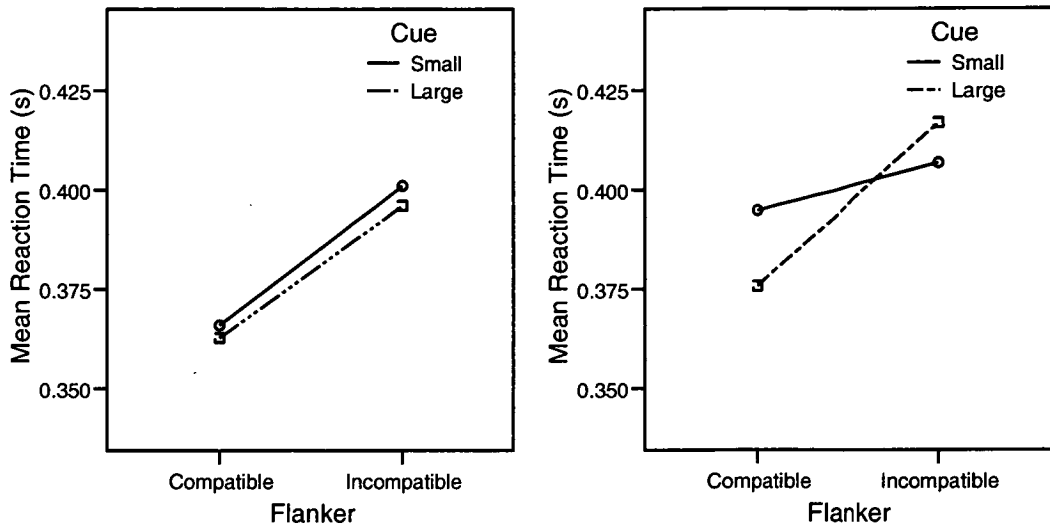


Figure 4. Mean RT for good decoders (left) and poor decoders (right) for the 0ms SOA (simultaneous) condition as a function of flanker compatibility and cue-size.

Figure 5 shows mean RT as a function of Cue, Group, and Flanker for the 500ms SOA condition. Good decoders showed a significant Cue x Flanker interaction, $F(1,15)=10.57$, $MSE=0.0001$, $p<.01$, indicating shorter RT for large in comparison to small cues for compatible stimuli, $F(1,15)=8.13$, $MSE=0.0002$, $p<.05$, but no effect of Cue for incompatible stimuli ($p>.05$). Poor decoders also showed a trend for a Cue x Flanker interaction, $F(1,12)=3.32$, $MSE=0.0007$, $p=.094$, indicating significantly longer RT for large in comparison to small cues for incompatible stimuli, $F(1,12)=10.53$, $MSE=0.0002$, $p<.01$, but the effect of Cue was non-significant for compatible stimuli ($p>.05$). Good decoders tended to show shorter RT in comparison to poor decoders for compatible stimuli preceded by large cues overall, $F(1,29)=3.45$, $MSE=.001$, $p=.073$.

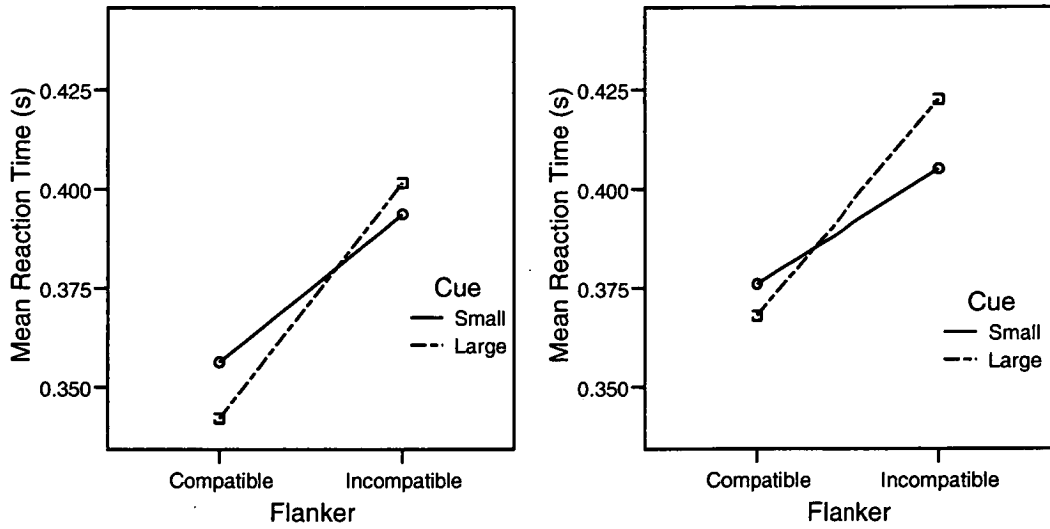


Figure 5. Mean RT for good decoders (left) and poor decoders (right) for the 500ms SOA (cued) condition as a function of flanker compatibility and cue-size.

Mean Percent Correct

Overall accuracy was greater for go ($M=98.6$, $SEM=0.309$) in comparison to nogo ($M=93.8$, $SEM=0.601$) trials, $F(1,27)=61.58$, $MSE=44.50$, $p<.001$, for the 500ms ($M=96.9$, $SEM=0.334$) in comparison to the 0ms SOA ($M=95.5$, $SEM=0.463$) condition, $F(1,27)=16.90$, $MSE=13.12$, $p<.001$, for large ($M=96.5$, $SEM=0.390$) in comparison to small ($M=95.9$, $SEM=0.403$) cues, $F(1,27)=5.29$, $MSE=10.41$, $p<.05$, and for compatible ($M=97.8$, $SEM=0.256$) in comparison to incompatible ($M=94.6$, $SEM=0.603$) stimuli, $F(1,27)=31.10$, $MSE=37.73$, $p<.001$. The main effect of Group was non-significant, $F(1,27)=0.068$, $MSE=65.0$, $p>.05$. Figure 6 shows a significant go/nogo x Flanker x Group interaction, $F(1,27)=5.53$, $MSE=26.41$, $p<.05$. The Flanker x Group interaction was significant for nogo stimuli, $F(1,27)=4.63$, $MSE=61.64$, $p<.05$, such that the percentage of correct trials was lower for incompatible than compatible stimuli for both good decoders, $F(1,15)=30.40$, $MSE=76.58$, $p<.001$, and poor decoders, $F(1,12)=10.43$, $MSE=1.88$, $p<.01$. Although this effect appears larger for good decoders, no between group differences were found to be statistically significant ($ps>.05$).

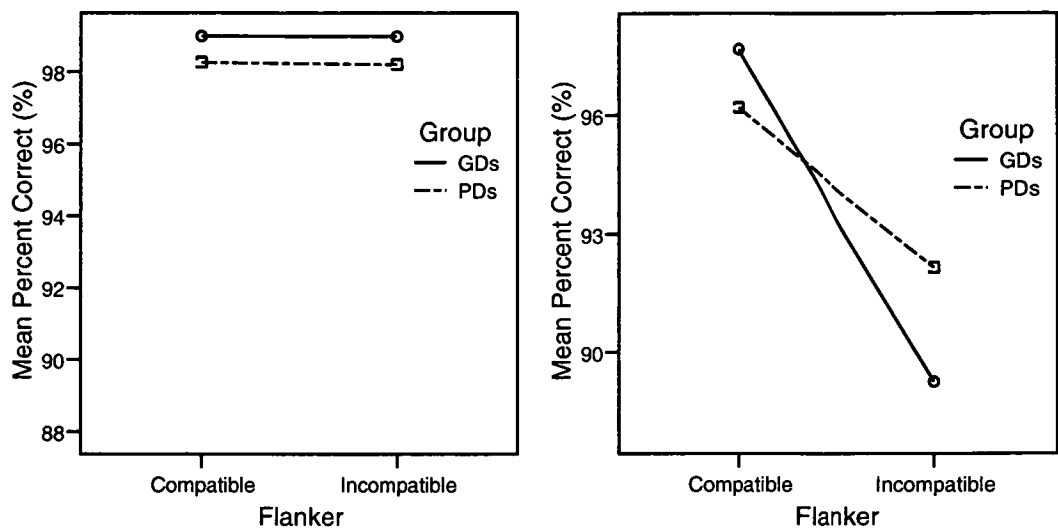


Figure 6. Mean percent correct for 'go' (left) and 'nogo' (right) trials as a function of flanker compatibility for good decoders (GDs) and poor decoders (PDs).

Electrophysiological Analyses

Figures 7 and 8 show grand mean averaged ERP waveforms for the simultaneous (0ms) and cued (500ms) conditions respectively at occipital (O1, O2), parietal (P3, P4), and temporal (P7, P8) sites as a function of Group, Flanker, and Cue. As expected, N1 amplitude appears greater for small relative to large cues overall, particularly for the 500ms SOA condition (Figure 8). An increase in N1 latency can also be observed for the 0ms SOA condition (Figure 7). The modulation of N1 amplitude by cue size appears to be greater in the LH for good relative to poor decoders, particularly at parietal (P3) and temporal (P7) for the 500ms condition. It is difficult to ascertain from these figures if there are group differences in the N1 amplitude for each type of stimulus. The following statistical analyses were performed to investigate these differences further.

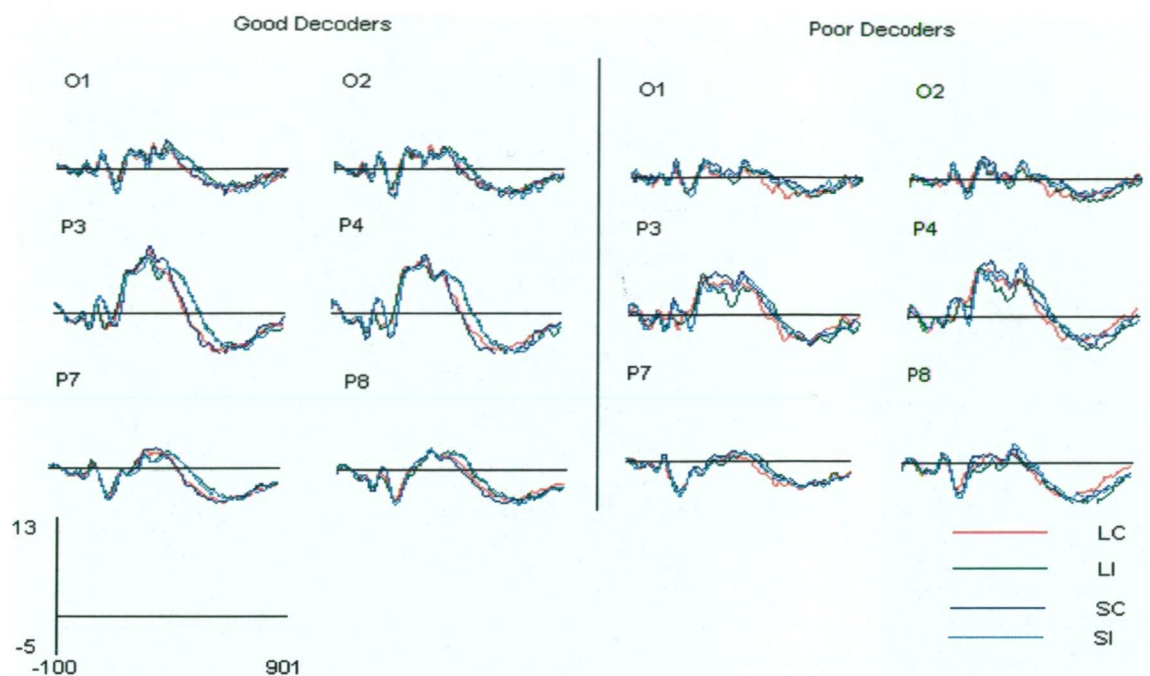


Figure 7. Grand mean averaged waveforms for good decoders (left) and poor decoders (right) at posterior sites for the 0ms SOA condition (LC=large cue-compatible; LI=large cue-incompatible SC=small cue-compatible; SI=small cue-incompatible).

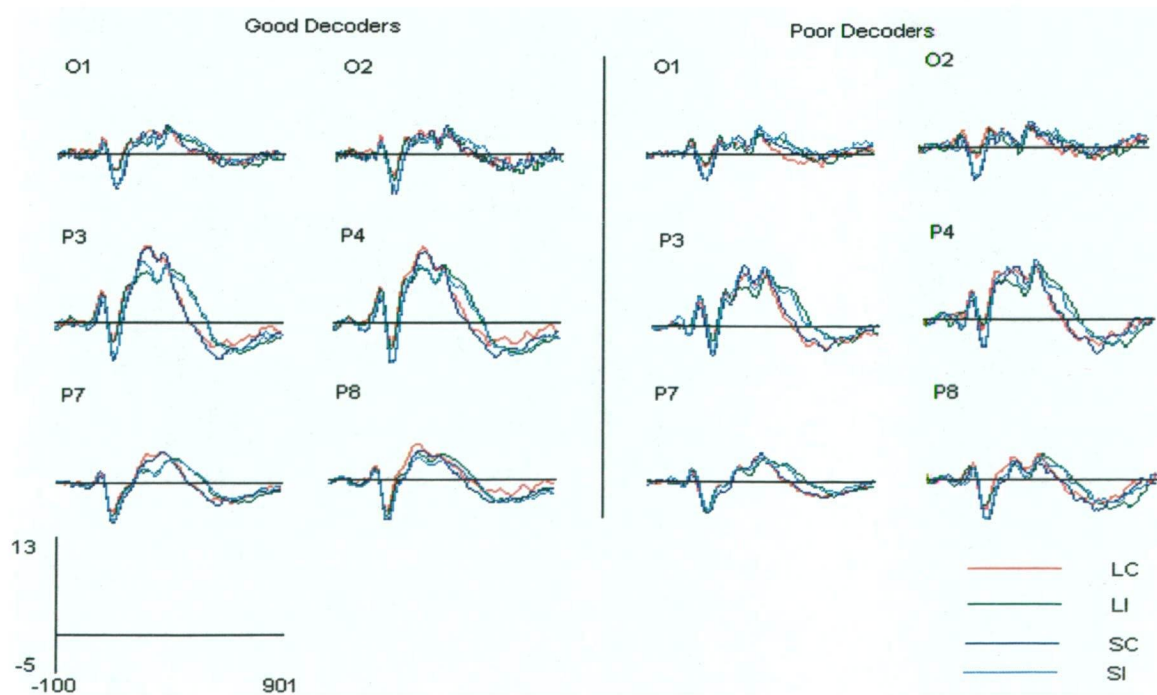


Figure 8. Grand mean averaged waveforms for good decoders (left) and poor decoders (right) at posterior sites for the 500ms SOA condition (LC=large cue-compatible; LI=large cue-incompatible SC=small cue-compatible; SI=small cue-incompatible).

Posterior N1 Amplitude

There was a significant main effect of Sagittal site, $F(2,54)=3.56$, $MSE=64.6$, $p<.05$, indicating significantly greater N1 amplitude at temporal ($M=-4.99$, $SEM=.263$) in comparison to occipital ($M=-3.72$, $SEM=.405$) sites, whereas N1 amplitude at parietal sites ($M=-4.46$, $SEM=.647$) was not significantly different to occipital or temporal sites. There was a significant main effect of Cue, $F(1,27)=14.82$, $MSE=18.35$, $p<.01$, indicating greater N1 amplitude for small ($M=-4.82$, $SEM=.446$) in comparison to large cues ($M=-3.95$, $SEM=.332$). However, this was modified by a significant Task x Cue interaction, $F(1,27)=7.34$, $MSE=15.10$, $p<.05$, such that the effect of Cue was significant for the 500ms SOA (cued) condition, $F(1,27)=21.17$, $MSE=17.24$, $p<.001$, but not for the 0ms SOA (simultaneous) condition ($p>.05$). This was further modified by a significant Sagittal x Task x Cue interaction, $F(2,54)=3.83$, $p<.05$. The overall effect of Cue was significant at each sagittal site, however, the Task x Cue interaction was significant at occipital, $F(1,27)=7.26$, $MSE=6.01$, $p<.05$, and parietal sites, $F(1,27)=6.56$, $MSE=14.64$, $p<.05$, such that the effect of Cue was significant for the 500ms but not the 0ms SOA condition.

Figure 9 shows a significant Cue x Hemisphere x Group interaction, $F(1,27)=5.58$, $MSE=3.67$, $p<.05$. The overall effect of Cue was significant for good decoders regardless of hemisphere, $F(1,15)=10.61$, $MSE=14.96$, $p<.01$. However, poor decoders showed a significant Cue x Hemisphere interaction, $F(1,12)=7.94$, $MSE=4.19$, $p<.05$, such that the effect of Cue was significant in the RH, $F(1,13)=9.57$, $MSE=0.883$, $p<.01$, but not the LH ($p>.05$). The main effect of Hemisphere was non-significant for both groups and there were no significant between group differences ($ps>.05$).

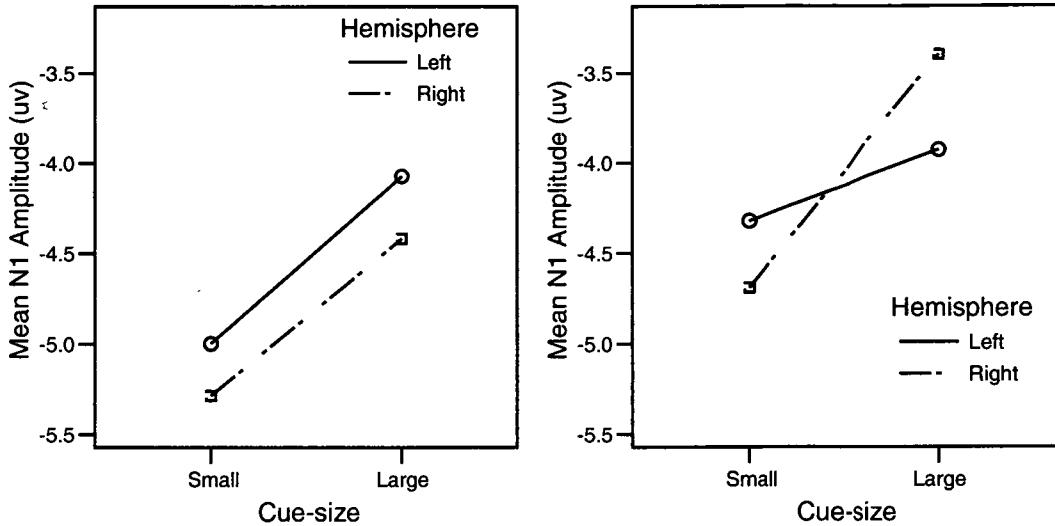


Figure 9. Mean N1 amplitude for good decoders (left) and poor decoders (right) as a function of Cue-size and Hemisphere.

There was a significant Task x Cue x Flanker x Hemisphere x Group interaction, $F(1,27)=7.69$, $MSE=1.211$, $p<.05$, such that for stimuli presented with large cues in the 0ms SOA (simultaneous) condition there was a significant Flanker x Hemisphere x Group interaction shown (see Figure 10), $F(1,27)=13.24$, $MSE=0.663$, $p<.001$. The Flanker x Hemisphere interaction was significant for good decoders, $F(1,16)=14.27$, $MSE=0.353$, $p<.01$, and poor decoders, $F(1,13)=4.89$, $MSE=0.419$, $p<.05$. Good decoders showed greater N1 amplitude in the RH for incompatible in comparison to compatible flankers, $F(1,16)=6.75$, $MSE=0.813$, $p<.05$, whereas poor decoders tended to show greater N1 amplitude in the LH for incompatible in comparison to compatible flankers, $F(1,13)=3.81$, $p=.073$. There was a significant Hemisphere x Group interaction for incompatible flankers preceded by large cues, $F(1,27)=4.99$, $MSE=3.44$, $p<.05$, such that good decoders tended to show greater N1 amplitude in the RH in comparison to poor decoders, $F(1,29)=5.02$, $MSE=5.03$, $p=.033$ ($p>.05$, Bonferroni corrected).

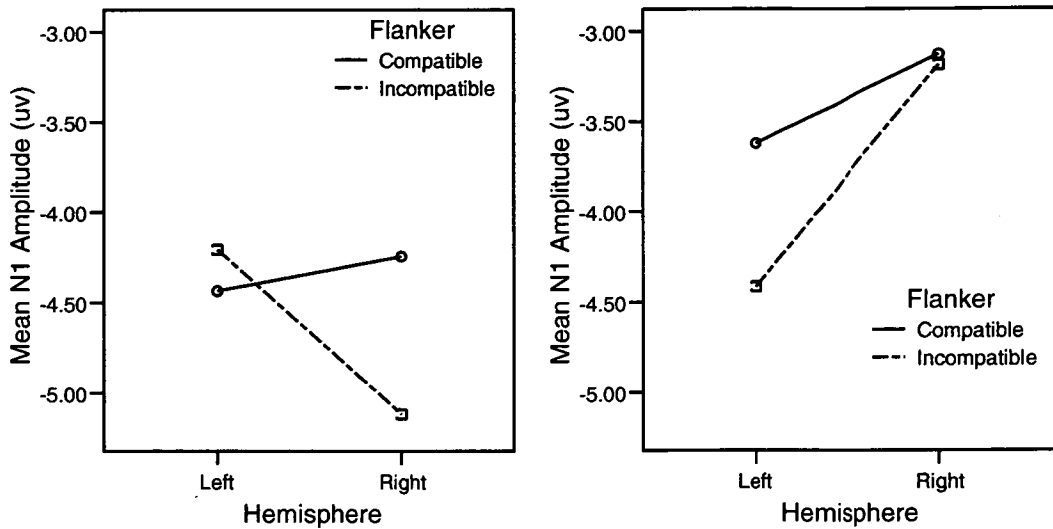


Figure 10. Mean N1 amplitude at posterior sites for good decoders (left) and poor decoders (right) for large cues in the 0ms condition as a function of hemisphere and flanker.

The Sagittal x Hemisphere x Group x Sex interaction was also significant, $F(2,54)=11.81$, $MSE=11.27$, $p<.001$, indicative of a significant Hemisphere x Group x Sex interaction at temporal sites (see Figure 11), $F(1,27)=11.41$, $MSE=30.77$, $p<.01$. Female PDs showed significantly greater N1 amplitude in the RH relative to the LH overall, $F(1,7)=8.89$, $MSE=3.68$, $p<.05$. Female GDs tended to show greater N1 amplitude in the LH in comparison to females PDs, $F(1,16)=3.57$, $MSE=2.13$, $p=.077$, and female PDs tended to show greater amplitude in the RH in comparison to female GDs, $F(1,16)=3.76$, $MSE=6.02$, $p=.07$. The Hemisphere x Group interaction approached significance for males, $F(1,11)=4.66$, $MSE=3.75$, $p=.054$, such that male GDs tended to show greater N1 amplitude in the RH in comparison to male PDs, $F(1,11)=4.44$, $MSE=3.56$, $p=.059$.

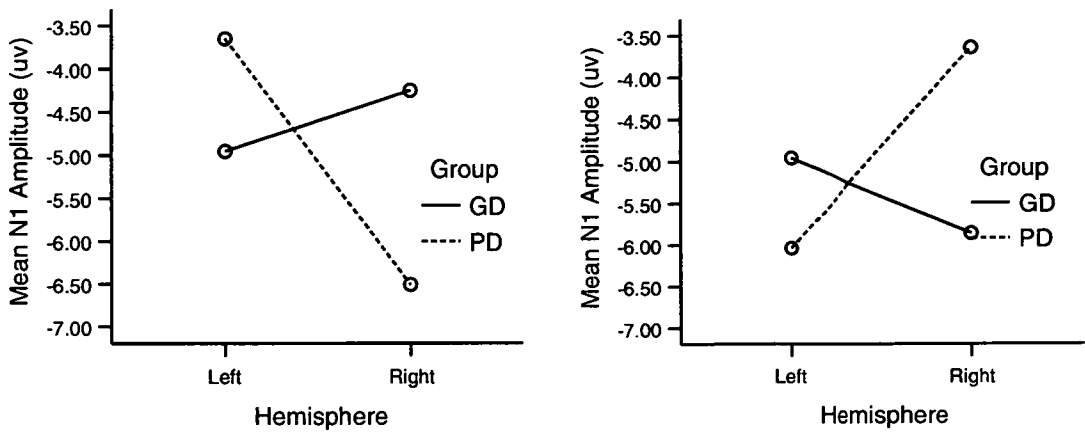


Figure 11. Mean N1 amplitude at LH and RH temporal sites for female (left) and Male (right) good decoders (GD) and poor decoders (PD).

Posterior N1 Latency

There was a significant main effect of Sagittal site, $F(2,54)=5.20$, $MSE=985.2$, $p<.01$, such that N1 latency was shorter at occipital ($M=154.04$, $SEM=2.15$) in comparison to parietal ($M=158.97$, $SEM=2.38$) and temporal sites ($M=160.11$, $SEM=1.86$) sites. The main effect of Cue was significant, $F(1,27)=37.79$, $MSE=388.4$, $p<.001$, indicating significantly greater N1 latency for small ($M=160.90$, $SEM=1.73$) in comparison to large ($M=154.51$, $SEM=2.02$) cues. There was a significant main effect of Flanker, $F(1,27)=6.14$, $MSE=269.7$, $p<.05$, indicating greater N1 latency for incompatible ($M=158.78$, $SEM=1.78$) in comparison to compatible ($M=156.63$, $SEM=1.93$) flankers. However, this was modified by a significant Flanker x Group interaction, $F(1,27)=5.03$, $p<.05$, such that the Flanker effect approached significance for poor decoders, $F(1,12)=4.80$, $p=.049$ ($p>.05$, Bonferroni corrected), and was non-significant for good decoders ($p>.05$).

Separate analyses were conducted for each SOA condition. Figure 11 shows a significant Cue x Hemisphere x Group interaction for the 500ms condition, $F(1,27)=5.23$, $MSE=56.22$, $p<.05$. The Hemisphere x Group interaction was significant for small cues, $F(1,27)=4.42$, $MSE=41.61$, $p<.05$, such that good decoders tended to show shorter N1 latency in

the RH in comparison to the LH, $F(1,15)=5.39$, $MSE=25.81$, $p=.035$ ($p>.05$, Bonferroni corrected), whereas there were no hemispheric effects for poor decoders and no significant between group differences ($ps>.05$). For good decoders, the effect of Cue was significant in the LH, $F(1,15)=12.50$, $MSE=16.64$, $p<.01$, and approached significance in the RH, $F(1,15)=4.46$, $MSE=18.64$, $p=.052$. However, for poor decoders the effect of Cue approached significance in the RH, $F(1,12)=4.10$, $MSE=47.97$, $p=.066$, and was non-significant in the LH ($p>.05$).

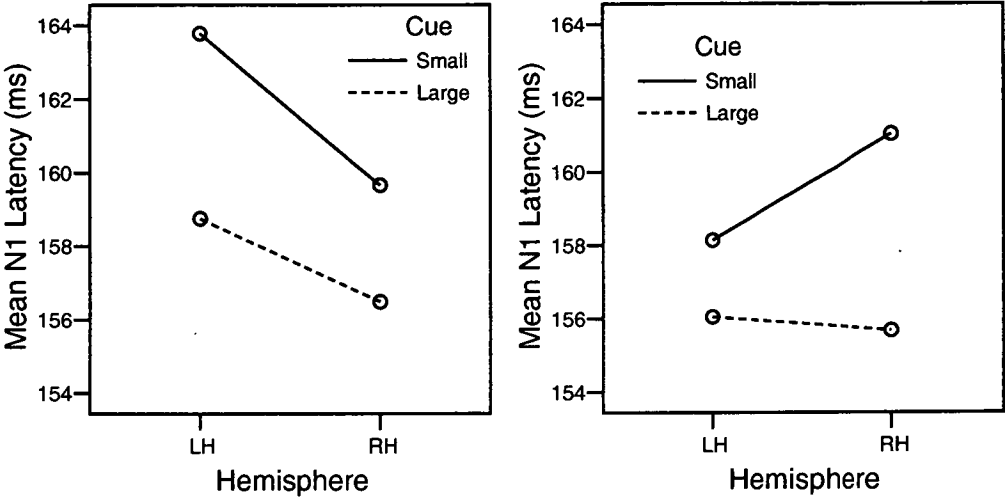


Figure 11. Mean N1 latency for the 500ms SOA condition as a function of Cue and Hemisphere for good decoders (left) and poor decoders (right).

Anterior N2 Amplitude

Figure 12 shows grand mean averaged waveforms at frontal sites for good decoders and poor decoders as a function of SOA, Cue, and Flanker. The ANOVA conducted on this data showed a significant main effect of Task, $F(1,27)=12.32$, $MSE=36.39$, $p<.01$, such that N2 amplitude was significantly greater overall for the 0ms SOA (simultaneous) condition ($M=-1.90$, $SEM=0.482$) in comparison to the 500ms SOA (cued) condition ($M=-0.32$, $SEM=0.470$). The main effect of Flanker was also significant, $F(1,27)=8.95$, $MSE=17.30$, $p<.01$, indicating greater N2 amplitude for incompatible ($M=-1.58$, $SEM=0.454$) in comparison to compatible ($M=-0.650$, $SEM=0.441$) flankers. There was also a significant main effect of Cue, $F(1,27)=8.03$, $MSE=9.08$,

$p < .01$, indicating greater N2 amplitude for large ($M = -1.43$, $SEM = 0.483$) in comparison to small ($M = -0.795$, $SEM = 0.379$) cues. The main effect of Group was non-significant ($p > .05$). The Flanker x Group interaction approached significance, $F(1,27) = 3.48$, $MSE = 17.29$, $p = .073$. Good decoders showed significantly greater N2 amplitude for incompatible in comparison to compatible flanker stimuli overall, $F(1,15) = 9.53$, $MSE = 23.53$, $p < .01$. However, the main effect of flanker was non significant for poor decoders and there were no significant difference between the groups for compatible or incompatible stimuli ($ps > .05$). This Flanker x Group interaction was found to be significant for the large cue condition, $F(1,27) = 4.97$, $MSE = 11.12$, $p < .05$, but not for the small cue condition ($p > .05$). There was also a trend for a Flanker x Coronal x Group interaction for large cues, $F(2,54) = 2.90$, $p = .064$, such that the Flanker x Group interaction was significant at F4 ($p < .05$), and approached significance at Fz ($p = .053$) and F3 ($p = .094$).

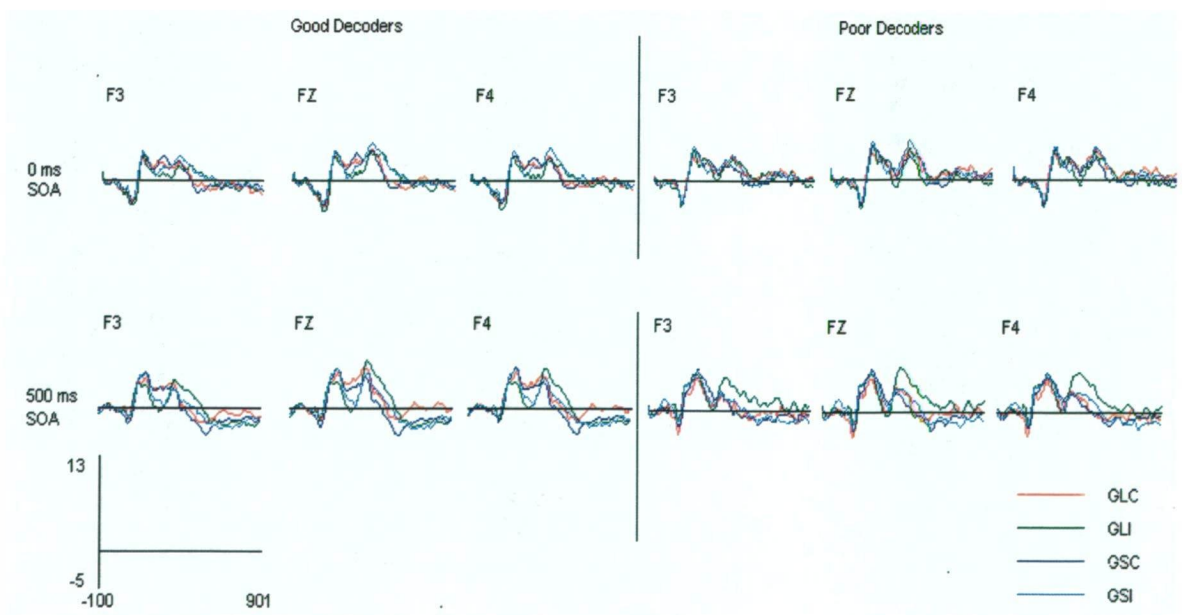


Figure 12. Grand mean averaged waveforms at frontal sites for good decoders (left) and poor decoders (right) for the 0ms SOA (top) and 500ms SOA (bottom) as a function of cue-size and flanker.

Anterior N2 latency

For N2 latency, the main effect of Flanker approached significance, $F(1,27)=4.04$, $MSE=1441$, $p=.055$, such that N2 latency tended to be shorter for consistent ($M=266.0$, $SEM=6.06$) in comparison to inconsistent ($M=271.7$, $SEM=5.41$) flankers. There were no other significant effects involving Flanker or Group ($ps>.05$).

Discussion

The behavioural data revealed significant flanker effects (Eriksen & Eriksen, 1974) and cue-size effects (e.g., Castiello & Umiltà, 1990) for both RT and accuracy. Mean RT was shorter and accuracy greater for the 500ms (cued) in comparison to the 0ms SOA (simultaneous) condition, indicating an additional RT benefit when the cue appeared prior to rather than simultaneously with the target stimulus. The effect of cue-size was found to interact with flanker compatibility. Small cues produced an additional RT benefit compared to large cues, when targets were flanked by incompatible flankers, suggesting that when the spatial scale of attention is smaller and more precise the effect of flanking stimuli is reduced (Eriksen & St James, 1986; Facoetti & Molteni, 2000). Consistent with previous findings (Callejas et al., 2004; Facoetti & Molteni, 2000), the flanker effect was found to be greater for large than small cues for both good and poor phonological decoders. However, this interacted with flanker compatibility differently for each group, particularly for the 500ms SOA condition during which attentional focussing was optimal. Under these conditions, good decoders showed an additional RT benefit for compatible stimuli preceded by large in comparison to small cues, but there was little effect of cue size for incompatible flankers. In contrast, poor decoders did not show the same advantage for compatible stimuli presented with large cues, but showed a greater RT cost of incompatible flankers when preceded by large cues. Broadening the scale of spatial attention increases flanker interference (Facoetti & Molteni, 2000) and the ability to ignore flankers is more difficult when the focus of attention needs to be shifted (Callejas et al., 2004). Thus, poor decoders were less

able to either focus spatial attention, or suppress information from flanker locations, under these conditions when compared to good decoders. Previous research findings indicate that dyslexics are also more receptive to flanker interference during the performance of similar tasks (Bednarek et al., 2004; Brosnan et al., 2002; Facoetti & Turatto, 2000; Klein & D'Entremont, 1999).

N1 amplitude at posterior sites was significantly greater for small in comparison to large cues indicating that it was modulated by the spatial scale of attention (Luo et al., 2001). As expected, this effect was greatest for the 500ms SOA (cued) condition when the effects of spatial attention were maximised. N1 latency was also significantly modulated by cue-size such that longer latency was observed for small in comparison to large cues which may reflect the time required to focus attention. Good and poor phonological decoders showed hemispheric differences in the modulation of N1 amplitude as a function of cue-size. Overall N1 amplitude was modulated by cue-size in both hemispheres for good decoders but only in the RH for poor decoders. This is consistent with hemispheric differences in the allocation of attentional resources in the LH, but is not consistent with the RH deficit that has been proposed to underlie attentional difficulties in dyslexia (Stein & Walsh, 1997). Similar hemispheric asymmetries were found for N1 latency in the cued condition. Good decoders showed significantly greater N1 latency in the LH for small in comparison to large cues and in comparison to the RH. In contrast the effect of cue approached significance in the RH for poor decoders, though this effect failed to reach conventional levels of statistical significance and should be interpreted with caution.

N1 amplitude was also modulated by flanker compatibility such that greater N1 amplitude was observed for incompatible relative to compatible flankers, particularly for the simultaneous condition. The modulation of N1 amplitude by flanker compatibility is consistent with the suggestion that N1 amplitude reflects the gradient of visual attention (Mangun & Hillyard, 1991) or the demands of visual discrimination within the focus of attention (Vogel & Luck, 2000). For stimuli presented with large cues under these conditions, good decoders showed significantly greater N1 amplitude in the RH for incompatible relative to compatible

flankers and this tended to be greater in comparison to poor decoders. In contrast, poor decoders tended to show a flanker effect in the LH but not the RH. These findings suggest that hemispheric differences in focussing the spatial scale of attention to letters in the presence of incompatible distractors underlie the greater flanker interference effect observed for poor decoders in the reaction time data. These findings are also consistent with the RH deficit hypothesis of dyslexia. In a recent behavioural study it was reported that normal readers showed a RH bias and adult dyslexics a LH bias in a visual spatial quantitative task, suggestive of parietal lobe dysfunction and a right-to-left shift in functional organisation (Boles & Turan, 2003).

The present study was not designed to investigate visual field differences, however, previous research has shown a reduction in the flanker effect in the LVF and strong flanker effect in the RVF among dyslexic children (Facoetti & Turatto, 2000). Patients with temporoparietal lesions show a reduction in flanker effect in the contralesional visual field, and a larger effect in the ipsilesional visual field (Ro et al., 1998), suggesting that dyslexics performance was due to a RH posterior deficit. Further, consistent with the magnocellular theory of dyslexia, there is evidence that the M system is involved in the allocation of attention during the identification of flanked-letter stimuli (Omtzigt & Hendriks, 2004; Omtzigt et al., 2002).

Although the N1 findings indicate differences in processing within the posterior parietal attentional network, there was also evidence for processing differences in the frontal attentional network. Consistent with previous research findings (Gratton et al., 1988; Heil et al., 2000; Kopp et al., 1996b) N2 amplitude at frontal sites was modulated by flanker compatibility reflecting the inhibition of irrelevant information by the frontal executive network. The flanker effect on N2 amplitude was significant for good decoders but not for poor decoders, particularly for stimuli preceded by large cues, and particularly at RH frontal sites. This finding suggests less inhibition of irrelevant flankers for poor phonological decoders and consistent with the greater flanker interference observed among this group in the RT data.

Among other areas, the anterior cingulate cortex and its interaction with the dorsolateral prefrontal cortex have been implicated in interference effects (Yamaguchi, Toyoda, Xu, Kobayashi, & Henik, 2002). The anterior cingulate is thought to monitor conflicting information and acts on the dorsolateral prefrontal cortex in order to increase selective attention to task relevant and suppress task irrelevant information. There is also some recent evidence the dorsal anterior cingulate is involved in boosting attention toward task relevant events as well as resolving conflict (Weissman et al., 2005). Based on Bakker's abnormal symmetry hypothesis, Brosnan et al. (2002) suggested that the difficulty that dyslexics show in inhibiting distracting information in an embedded figures test could be related to a deficit in the left prefrontal region that is responsible for inhibiting the processing of the right prefrontal region. Bednarek et al. (2004) also suggested that the behavioural impairment exhibited by dyslexics when targets were flanked by incompatible flankers was suggestive of a deficit in executive control rather than orienting or alerting (see also Kelly, Best, & Kirk, 1989, for a prefrontal cortical hypothesis of dyslexia).

For compatible stimuli, large cues tended to produce an additional RT benefit relative to small cues overall, particularly in the cued condition, and this was greater for good relative to poor decoders. Further, when the cue and target appeared simultaneously, good decoders showed a flanker effect but cue-size had little impact on RT, which is consistent with previous research showing a reduction in cue-size effects at short SOAs (Benso et al., 1998; Castiello & Umiltà, 1990). In contrast, poor decoders showed a flanker effect when the entire stimulus was surrounded by the cue (large cue), but not when just the middle target letter of the stimulus was surrounded by the cue (small cue). This effect was largely due to increased RT for compatible flanker stimuli presented simultaneously with a small cue. Together these findings show that poor decoders benefited less from priming of target representations by response compatible flankers.

There are several possible explanations for the greater facilitation effects observed for good decoders in response to compatible flankers. Previous research findings also indicate a benefit of compatible flankers relative to baseline conditions (Eriksen & Eriksen, 1974) and some researchers have shown repetition priming effects when targets are preceded by compatible flankers (Flowers, 1990; Flowers & Wilcox, 1982). For poor decoders, the information gathered at an early parallel processing stage may have been insufficient for the discrimination of targets and flankers. Based on the effects of expectancy on the flanker effect, it has been argued that each hemisphere may be biased towards giving more or less weight to information extracted during early visual processing (Corballis & Gratton, 2003). Information gained in the early parallel processing stage may in turn affect the ability to maintain a wide or narrow attentional focus in the case of compatible and incompatible stimuli respectively, and may also influence inhibitory processing in the frontal attentional network.

It has also been suggested that congruence effects occur due to feature integration and perceptual grouping strategies that are developed for encoding graphical symbols (van Leeuwen & Lachmann, 2004). The facilitation or suppression of feature integration may depend on whether an analytic (local) or holistic (global) strategy is used. If the stimulus is processed holistically as a shape, targets and distractors are integrated (at either the phonological or abstract letter level), leading to positive congruence effects. However, if the preferred strategy is letter perception, global features are suppressed in favour of abstract identity codes and negative flanker effects may occur such that a cost is observed when flankers are compatible with the target (van Leeuwen & Lachmann, 2004). The perceptual integration of shapes and the non-integration of letter processing may run in parallel in different hemispheres, with one receiving priority over the other depending on the domain (Bedson & Turnbull, 2002). A bias towards global processing could result in increased processing of flanking information and therefore a larger interference effect or a greater benefit of compatible flankers (Corballis & Gratton, 2003). Previous research suggests that the RH and LH preferentially process global and local

information respectively (Fink, Marshall, Halligan, & Dolan, 1999; Martinez et al., 1997; Weber, Schwarz, Kneifel, Treyer, & Buck, 2000; Yamaguchi, Yamagata, & Kobayashi, 2000). Thus it is possible that the modulation of N1 amplitude by flanker compatibility in the RH for good decoders and the LH for poor decoders indicates a bias towards global and local processing strategies respectively.

According to the type token individuation hypothesis, letters can be represented as either 'types' that code abstract categorical features, or 'tokens' that register shape information. Repetition is thought to increase type-level and decrease token-level information in target detection tasks such as those used in the present study. Rouder and King (2003) found negative flanker effects when targets were surrounded by morphed letters, suggesting that 'type' information is used for well formed letters and 'token' level information for morphed or masked letters. Thus the greater benefit of compatible flankers observed for good decoders may reflect greater ability to use type level information. Kanwisher (1991) suggested that word recognition can proceed through either a fast types-first route or a slower and more attentionally demanding tokens-first route. These two routes correspond to the reading styles of L-type and P-type dyslexia, such that P-type dyslexics have difficulty identifying similar sounding letters and L-type dyslexics confuse letters with similar shapes (Mather, 2001). It has further been suggested that neural pathways for processing type and token information correspond to the ventral (occipito-temporal) and dorsal (occipito-parietal) visual processing pathways respectively, such that the ventral stream codes distinctive features or attributes, whereas the dorsal stream codes shape and/or location information. These two pathways are thought to converge on the LH angular gyrus which is involved in conjoining these two sources of information.

Although the present study was not designed to investigate sex differences, there was evidence for an overall reversal in hemispheric asymmetries for males and females at temporal sites that interacted with decoding group. Female poor decoders showed a reduction in N1 amplitude in the LH relative to female good decoders, which is consistent with the LH deficit

hypothesis of dyslexia. However, female poor decoders showed greater amplitude in the RH relative to female good decoders which indicates compensatory processing. This is consistent with evidence for compensatory processing in the RH in dyslexia. In contrast, male poor decoders showed a reduction in N1 amplitude in the RH relative to male good decoders which is consistent with the RH deficit hypothesis of dyslexia. These findings also indicate that female good decoders were more likely to use a LH (linguistic) strategy, whereas males were more likely to use a RH (spatial) strategy which is consistent with previously reported sex differences in hemispheric processing (Gur et al., 2000). These findings should be considered preliminary but suggest that sex differences in hemispheric asymmetries may contribute to the attentional and reading difficulties experienced by poor phonological decoders and possibly dyslexics.

In summary, poor decoders showed a greater RT cost for incompatible stimuli preceded by large cues indicating differences in focussing the spatial scale of attention and suppressing information at unattended locations. This is consistent with the suggestion that the inhibition of distractors is important for successful reading (LaBerge & Brown, 1989) and for developing grapheme phoneme conversion strategies (Brosnan et al., 2002). Together the N1 findings suggest hemispheric differences in both attentional focussing and cue-driven attentional shifting. In particular the greater interference effects observed for poor decoders in the RT data was accompanied by a reduction in N1 modulation in the RH which is consistent with the RH deficit hypothesis of dyslexia. Attentional shifting and attentional focussing are partially independent but are both mediated by the posterior attentional network (Posner & Peterson, 1990). However there was also evidence for differences in processing in the frontal attentional network, as indexed by the frontal N2 component. Good decoders also benefited more from compatible flanker information which could reflect group differences in global/local processing strategies. Thus the following experiment was conducted to examine differences between good and poor phonological decoders in the allocation of attention to global and local levels of hierarchical stimuli.

CHAPTER 8 - EXPERIMENT 3: ELECTROPHYSIOLOGICAL INDICES OF GLOBAL/LOCAL PROCESSING IN GOOD AND POOR PHONOLOGICAL DECODERS

Reading requires the allocation of attention to both individual letters and whole words comprised of individual elements and therefore requires both global (holistic) and local (analytic) visual processing strategies. The findings of studies employing a variety of paradigms indicate that poor readers are inclined towards more diffuse allocation of attention or an inclination for global processing (Facoetti & Molteni, 2001; Facoetti et al., 2000a; von Karolyi, 2001; von Karolyi et al., 2003; Williams & Bologna, 1985). Facoetti et al. (2000) found that dyslexic children showed a smaller increase in RT relative to controls for dot detection and visual search tasks as a function of target eccentricity and number of distractors respectively. This suggested difficulty in narrowing the focus of attention and inhibiting laterally distracting information resulting in a tendency to analyse visual patterns by the distributed mode of visual attention. Other research suggests that dyslexics are better than controls at recognising impossible figures (a global visual-spatial task) suggesting enhanced ability to process information in a global or holistic fashion (von Karolyi, 2001; von Karolyi et al., 2003). Williams and Bologna (1985) found that poor readers showed greater perceptual grouping effects in a selective attention task (speeded card sorting) than good readers suggestive of an inclination for holistic or rather than analytic local processing (Williams & Bologna, 1985).

Global and local processing is often investigated using hierarchical stimuli consisting of an overall global letter that is composed of local letters that are either consistent or inconsistent with the global form (Navon, 1977). A RT advantage is typically observed for the selection of global features when compared to local features (global precedence effect), and a global-to-local interference effect is observed when the overall global feature is inconsistent with the local feature (for a review see Kimchi, 1992). The global precedence effect was initially proposed to reflect the order of processing within the visual system (Navon, 1977), but further research has shown that global and local levels are processed in parallel and that both perceptual and

attentional mechanisms contribute to the global precedence effect (Hübner & Malinowski, 2002). A relationship has been demonstrated between global/local processing and spatial frequency, such that global processing is associated with the perception of low spatial frequencies and local processing with high spatial frequencies (Badcock et al., 1990; Shulman & Wilson, 1987a). These findings implicate the rapid low frequency magnocellular visual processing system and the slower medium to high frequency parvocellular system in global and local processing respectively (Badcock et al., 1990).

Attentional factors have been shown to moderate the global precedence effect (Hübner, 2000; Stöffer, 1994; Weber et al., 2000). The allocation or shifting of attention is affected by both bottom-up (stimulus driven) and top-down (goal directed) processes (Johnston & Dark, 1986) or exogenous and endogenous modes of attention (Posner, 1980). When hierarchical stimuli are preceded by spatial cues that draw attention to the local level there is a reduction in the global precedence effect and global-to-local interference (Han & He, 2003) indicating that top-down voluntary attentional control can overcome exogenous attentional capture (Hübner, 2000; Stöffer, 1994). According to attentional zooming theory (Eriksen & St James, 1986), the abrupt stimulus onset of hierarchical stimuli captures attention to the global level and the global precedence effect reflects the time required to refocus attention to the local level (Stöffer, 1994). Thus, global processing may be likened to an expansion of the spotlight/zoom lens, whereas local processing indicates a narrowing of the attentional focus. This is supported by findings of an MRI and eye movement study which demonstrated activation of oculomotor areas (often resulting in saccades) during local processing suggesting that attending to local details induces a shift or narrowing of attention, whereas attending to global features induces an expansion of attention (Weber et al., 2000).

Few previous studies have investigated the relationship between global/local processing and reading ability using traditional hierarchical stimuli. Williams and LeCluyse (1990) cite an in press publication of Williams and Brannan in which there was no effect of consistency for

local letter identification among disabled readers. However, when the stimulus was blurred, the RT of disabled readers became faster and the consistency effect appeared. This was argued to occur due to re-establishment of the temporal relationship between the transient and sustained systems. Keen and Lovegrove (2000) investigated the effects of size and retinal eccentricity on global/local processing in dyslexic children and chronological and reading age matched controls. Considering that dyslexia has been proposed to be associated with a deficit in the magnocellular visual processing stream it was argued that this would transfer to a deficit in global in comparison to local processing. However, whereas dyslexics showed longer RT in comparison to chronological age matched controls and shorter RT in comparison to reading age matched controls, there were no differences in global precedence or eccentricity effects. Thus, it was argued that dyslexics are slow at processing visual information which affects the rapid processing of peripheral information and its integration across fixations during reading. However, another possibility is that the overall performance decrement was due to a deficiency in allocating spatial attention to either the global or local level.

Global/Local processing is associated with attentional control by temporo-parietal areas over earlier processing in the visual cortex (Fink et al., 1999). Several electrophysiological studies have demonstrated task and attention related modulation of posterior ERP components during global/local processing (Han & He, 2003; Han, Liu, Yund, & Woods, 2000; Heinze, Hinrichs, Scholz, Burchert, & Mangun, 1998). Modulation of the N1 component has been shown when attention is sustained to either the global or local level of hierarchical stimuli, such that N1 amplitude is greater at attended in comparison to unattended locations and greater when attention is sustained at the local level in comparison to the global level (Han et al., 2000). This is consistent with the body of electrophysiological research showing that the N1 component is modulated by spatial attention (Eimer, 1998; Mangun, 1995), and provides further support for the relationship between global/local processing and spatial attention.

The N2 component is also modulated by global/local processing level such that greater amplitude is observed for local in comparison to global features under conditions of selective (Han & He, 2003; Han et al., 2000; Heinze et al., 1998) but not divided attention (Heinze et al., 1998). The reduction in the global precedence effect when a pop-out target was presented among local letters resulted in decreased posterior N2 and increased anterior N2 amplitude reflecting separate anterior and posterior attentional networks (Han & He, 2003).

The aim of the present study was to investigate the relationship between electrophysiological measures of spatial attention and global/local processing in good and poor adult phonological decoders. If poor phonological decoders show a selective deficit in global processing this would indicate difficulty in low spatial frequency information processing and be consistent with a magnocellular deficit. However, if poor decoders show an advantage for the global task this would indicate a propensity to process visual stimuli in a global or holistic fashion. Further, an overall decrement in performance relative to good decoders would be consistent with difficulty in focussing and expanding the spatial scale of attention. The modulation of the posterior N1 component was examined as an index of processing within the focus of attention and the posterior rather than anterior N2 was examined to further investigate processing within the posterior attentional network. Good decoders are expected to show greater N1 and N2 amplitude for the local in comparison to the global task. If poor readers are less efficient in changing the focus of attention as a function of task demands they are not expected to show the same task-related modulation of these ERP components.

Method

Participants

This study was approved the University of Tasmania Human Research Ethics Committee. Thirty-six first year psychology students at the University of Tasmania participated in the experiment as part of their course requirement and all gave written informed consent prior to participation. Exclusion criteria included a history of drug, alcohol, or tobacco abuse, psychiatric or neurological disorder, head trauma, seizure, and those currently receiving medication. Good ($n=18$) and poor ($n=17$) phonological decoders were selected from a sample of over 300 Psychology 1 students on the basis of Martin and Pratt Nonword Reading Test scores (Martin & Pratt, 2001). Two good (1M, 1F) and two poor (2F) phonological decoders were left handed, the remaining participants were right handed as measured by the Edinburgh handedness inventory (Oldfield, 1971). All participants had normal or corrected to normal vision. One female poor phonological decoder was excluded from analyses due to outlying accuracy scores. The remaining sample consisted of 18 good (11F, 7M) and 16 (10F, 6M) poor decoders. The scores of good phonological decoders ranged from 49-54 (out of a possible score of 54) and the scores of poor phonological decoders ranged from 19-43. Norms for the Nonword reading test are available for samples up to 17 years of age (Martin & Pratt, 2001). The mean score of good decoder group was in the 81st percentile (> 17 years reading age equivalent) and the mean score of poor decoders was in the 19% percentile (10-11 years reading age equivalent) of this norming group.

Several reading and neuropsychological measures were administered in a screening session of approximately one hour on a day prior to the experimental session. Ravens Advanced Progressive Matrices (APM) was administered as a measure of non-verbal general intelligence (Raven et al., 1994). Other reading measures included the Word Identification and Comprehension subtests from the Woodcock-Johnson (WJ) Reading Mastery Tests (Woodcock, 1987), The National Adult Reading Test (NART: Nelson & Willison, 1991), an irregular word

reading test (see Appendix A), and Reading Accuracy and Reading Rate measures from the Neale Analysis of Reading Ability. Other measures included the Digit Span, Vocabulary, Symbol Coding and Symbol Copy sub-tests from the Weschler Adult Intelligence Scale (WAIS-III: Wechsler, 1997).

Table 4 shows mean ages and raw scores on each of these measures for each group. No between group differences were found in terms of age, general nonverbal intelligence (APM) and Digit Span Backwards. Good decoders were found to have significantly higher scores in comparison to poor decoders on reading and other cognitive measures including: non-word reading, word identification, irregular word reading, passage comprehension, vocabulary, reading rate, reading accuracy, symbol coding and symbol copy, and digit span forwards. A ceiling effect was observed for non-word reading scores, as the upper range for inclusion into the study was much smaller than the lower range to ensure large differences between groups. There were no significant sex effects or interactions involving sex for any measure.

Stimuli and Apparatus

Stimuli were presented on an IBM computer and tasks were programmed using the NeuroScan STIM program. Each compound stimulus consisted of a 3.5cm x 5.5cm global letter (E or H), which was comprised of 25 (E) or 19 (H) local elements. Local elements were 0.4cm x 0.6cm in size. The local letter stimuli were either the same as (consistent) or different (inconsistent) to the global letter. The inner edge of each compound letter stimuli was presented 1cm from fixation. A central fixation cross remained on the screen throughout stimulus presentation. Each block consisted of 100 trials, and stimuli appeared randomly in the LVF or RVF on 80% of trials. The remaining trials (20%) were catch trials in which a letter was presented in both visual fields simultaneously. The SOA between successive trials within each block varied randomly between 600-999ms.

Table 4.

Mean age and raw scores on reading and cognitive measures for good and poor phonological decoders in Experiment 3.

	Good Decoders		Poor Decoders		Sig.
	(n=18)		(n=16)		
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	
Age	21.47	3.80	19.71	2.27	<i>ns</i>
APM /36	23.67	5.03	21.50	3.61	<i>ns</i>
MP Nonword Reading /54	50.72	1.49	35.50	6.55	***
WJ Word Identification /106	98.50	2.57	87.63	4.91	***
Irregular word reading /87	78.39	8.63	61.38	7.58	***
NART [#] /50	34.06	4.19	18.56	4.44	***
Digit Span Forwards [#] /16	11.72	1.67	9.13	2.25	***
Digit Span Backwards [#] /14	7.94	1.55	7.25	2.14	<i>ns</i>
WJ Comprehension [†] /68	60.89	2.63	56.13	4.41	**
WAIS Vocabulary [†] /66	55.1	5.2	43.0	7.2	***
WAIS Symbol Coding [†] /60	81.3	8.3	74.3	7.9	*
WAIS Symbol Copy [†] /133	126.0	10.2	114.6	17.7	*
Neale Reading Accuracy [†] (%)	98.3	2.1	87.9	5.3	***
Neale Reading Rate [†] (words/min)	148.9	16.8	110.5	10.9	***

Note: * $p < .05$, ** $p < .01$, *** $p < .001$, [#] Missing data was substituted with the mean for the group for two poor decoders and two good decoders, [†] Missing data was substituted with the mean for the group for five good decoders and three poor decoders.

Electrophysiological Recording

EEG activity was recorded with a NeuroScan system, consisting of a 32-channel Synamps, SCAN 4.1 software, and Quik-cap with Ag/AgCl electrodes interfaced with a NeuroScan STIM 3.1 computer. EEG was recorded from 32 sites, according to the international 10-20 system, and all electrodes were referenced to the mastoids. Horizontal electro-oculographic (EOG) activity was recorded bipolarly from electrodes at the outer canthi of both eyes, and vertical EOG was recorded from electrodes above and below the left eye. Electrode impedance was kept below 5 k Ω . EEG activity was amplified with a band pass of 0.15-100 Hz and sampled continuously at a rate of 1000 Hz. Continuous EEG files were merged with behavioural files and band-pass filtered (0.15-30Hz, 24dB rolloff). Ocular artifact reduction was performed by regression and artifact averaging (Semlitsch, Anderer, Schuster, and Presslich, 1986). Data files were epoched offline for a 1000ms epoch commencing 100ms before stimulus onset and were baseline corrected at the pre-stimulus interval. High and low voltage cut-offs for artifact rejection were 100 μ V and -100 μ V respectively. Correct responses were averaged for each stimulus type and band-pass filtered (0.15-30 Hz). ERP components at posterior sites were determined from grand averaged means as the maximum voltage within the following time frames after target stimulus onset: N1 (130-190ms), N2 (230-290ms).

Procedure

Following set-up for EEG recording, participants were seated in front of a computer monitor; at a viewing distance of 70cm. Participants were given a verbal explanation of the task and completed 20 practice trials. The global and local tasks were performed in counterbalanced order. Within each of these tasks four blocks were completed in counterbalanced order, one for each letter (E and H) and each visual field (LVF, RVF) combination. Each block began with a written instruction (e.g., In this task you are asked to respond when you see a global H on the right. An example will follow), followed by an appropriate example of a target stimulus.

Participants were required to respond with a right hand button press whenever the attended letter (E or H) appeared on the attended side (LVF or RVF). Participants were instructed to respond as quickly and as accurately as possible to all target stimuli and to avoid blinking or overtly moving their head or eyes from central fixation. Participants took short breaks between tasks to prevent fatigue. The experimental session lasted approximately two hours (including set up for electrophysiological recording) with most participants completing several different experiments within the session.

Design and Data Analysis

Mean reaction time, accuracy, and mean amplitude and latency of ERP components were averaged across letter (E and H). Only data for correct responses to target stimuli were analysed. The effects of experimental manipulations on mean RT and accuracy were investigated using 2[Group: good decoder, poor decoder] x 2 [Sex: male, female] x 2(Task: global, local) x 2(Visual field: LVF, RVF) x 2(Consistency: consistent, inconsistent) repeated measures ANOVAs with two between subjects factors. The effects of experimental manipulations on mean N1 and N2 amplitude and latency were analysed by including the additional factors of (Sagittal site: occipital, temporal, parietal) and (Hemisphere: left, right) or (Laterality: contralateral, ipsilateral). All analyses were conducted in SPSS with Greenhouse-Geisser corrections where appropriate. Significant interactions were further investigated using breakdown ANOVAs with Bonferroni adjusted p-values for analysis of simple effects. Although Sex was included as a factor in all analyses only effects in which Group interacted with Sex are reported.

Results

Mean Reaction Time

Overall mean RT (s) was significantly greater for the local ($M=.407$, $SEM=.006$) in comparison to the global ($M=.370$, $SEM=.006$) task, $F(1,30)=57.90$, $MSE=.002$, $p<.001$, for LVF ($M=.395$, $SEM=.006$) in comparison to RVF ($M=.383$, $SEM=.006$) trials, $F(1,30)=17.42$, $MSE=.001$, $p<.001$, and for inconsistent ($M=.401$, $SEM=.006$) in comparison to consistent ($M=.377$, $SEM=.006$) stimuli, $F(1,30)=49.87$, $MSE=.001$, $p<.001$. The Task x Consistency interaction was significant, $F(1,30)=40.75$, $MSE=.0005$, $p<.001$, such that the effect of Consistency was significant for the local, $F(1,30)=80.75$, $MSE=.001$, $p<.001$, but not the global task ($p>.05$). Overall mean RT was significantly greater for poor decoders ($M=.401$, $SEM=.008$) in comparison to good decoders ($M=.376$, $SEM=.007$), $F(1,30)=5.01$, $MSE=.008$, $p<.05$. There were no significant higher order interactions involving group. There was a significant Task x Sex interaction, $F(1,30)=4.31$, $p<.05$. Females tended to show longer RT in comparison to males for the global task, $F(1,30)=5.30$, $MSE=.004$, $p=.028$ ($p>.05$, Bonferroni corrected).

Mean Accuracy

The percentage of correct trials was significantly lower for the local ($M=97\%$, $SEM=.582$) in comparison to the global ($M=98\%$, $SEM=.006$) task, $F(1,30)=5.33$, $MSE=25.71$, $p<.05$, and for inconsistent ($M=97\%$, $SEM=.565$) in comparison to consistent ($M=98\%$, $SEM=.411$) stimuli, $F(1,30)=6.35$, $MSE=26.19$, $p<.05$. There was a significant Task x Consistency interaction, $F(1,30)=25.57$, $MSE=8.89$, $p<.001$. The effect of consistency was significant for the local task, $F(1,30)=21.84$, $MSE=17.91$, $p<.001$, but not the global task ($p>.05$). Planned comparisons were performed to investigate differences between good and poor decoders for the global and local tasks separately. Poor decoders showed significantly lower accuracy ($M=97\%$, $SEM=.520$) in comparison to good decoders ($M=99\%$, $SEM=.556$) for the

global task, $F(1,30)=6.51$, $MSE=18.53$, $p<.05$, and there was no significant difference between the groups for the local task ($p>.05$).

Electrophysiological Data

As decoding group did not interact with consistency in the behavioural data, electrophysiological analyses were averaged across stimulus consistency. Figure 13 shows grand mean averaged ERP waveforms for good decoders and poor decoders for the global and local tasks. At parietal sites in both the LH (P3) and RH (P4), good but not poor decoders show greater N1 amplitude for the local relative to the global tasks. In contrast, poor decoders seem to show greater negativity at posterior sites within the N2 timeframe.

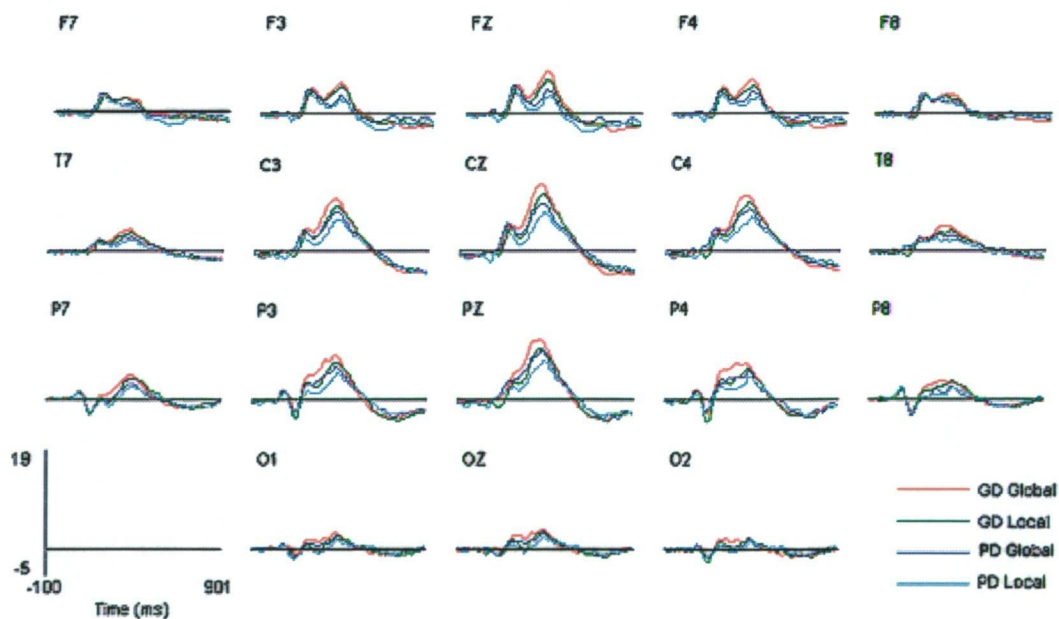


Figure 13. Grand mean averaged ERP waveforms for the global and local task for good and poor phonological decoders.

N1 Amplitude

N1 amplitude was greater at parietal ($M=-4.60$, $SEM=0.55$) in comparison to occipital ($M=-3.08$, $SEM=0.37$) sites, and tended to be greater at parietal in comparison to temporal ($M=-3.62$, $SEM=0.26$) sites, $F(2,60)=7.45$, $MSE=22.99$, $p<.01$. N1 amplitude was significantly greater for LVF ($M=-4.05$, $SEM=0.38$) in comparison to RVF ($M=-3.48$, $SEM=0.33$) trials $F(1,30)=5.69$, $MSE=11.14$, $p<.05$, and contralateral ($M=-5.18$, $SEM=0.42$) in comparison to ipsilateral ($M=-2.35$, $SEM=0.31$) sites overall, $F(1,30)=87.90$, $MSE=17.41$, $p<.001$. The main effect of Hemisphere approached significance such that N1 amplitude tended to be greater in the RH ($M=-4.07$, $SEM=0.37$) in comparison to the LH ($M=-3.47$, $SEM=0.38$), $F(1,30)=3.70$, $MSE=18.71$, $p=.064$.

N1 amplitude was significantly greater for the local ($M=-4.03$, $SEM=0.36$) in comparison to the global ($M=-3.51$, $SEM=0.35$) task overall, $F(1,30)=5.96$, $MSE=8.67$, $p<.05$, but this was modified by a significant Task x Sagittal site interaction, $F(2,60)=6.32$, $MSE=1.90$, $p<.01$. The effect of Task was significant at occipital, $F(1,30)=6.66$, $MSE=2.33$, $p<.05$, and parietal, $F(1,30)=7.09$, $MSE=7.60$, $p<.05$, but not at temporal sites ($p>.05$). There was a significant Task x Group interaction, $F(1,30)=4.21$, $MSE=8.67$, $p<.05$. Good decoders showed significantly greater N1 amplitude overall for the local in comparison to the global task, $F(1,16)=8.82$, $MSE=10.63$, $p<.01$. The effect of Task was non-significant for poor decoders, and there was no overall group difference for either task ($ps>.05$).

There was a significant Sagittal x Hemisphere x Group x Sex interaction, $F(2,60)=6.76$, $MSE=5.93$, $p<.01$, such that the Hemisphere x Group x Sex interaction was significant at temporal sites, $F(1,30)=8.43$, $MSE=5.93$, $p<.01$ (Figure 14), but not at parietal or occipital sites ($ps>.05$). At temporal sites, female poor decoders showed significantly greater N1 amplitude in the RH in comparison to the LH, $F(1,10)=6.48$, $MSE=4.10$, $p<.05$, and this tended to be greater in comparison to female good decoders, $F(1,19)=4.39$, $MSE=5.58$, $p=.05$ ($p>.05$, Bonferroni corrected), and male poor decoders, $F(1,14)=5.30$, $MSE=5.13$, $p=.037$ ($p>.05$, Bonferroni

corrected). Female good decoders and male poor decoders did not show any significant hemispheric differences ($ps>.05$). Male good decoders showed significantly greater N1 amplitude in the RH in comparison to the LH, $F(1,6)=6.75$, $MSE=0.97$, $p<.05$, and this tended to be greater in comparison to male poor decoders, $F(1,11)=5.99$, $MSE=1.49$, $p=.032$ ($p>.05$, Bonferroni corrected).

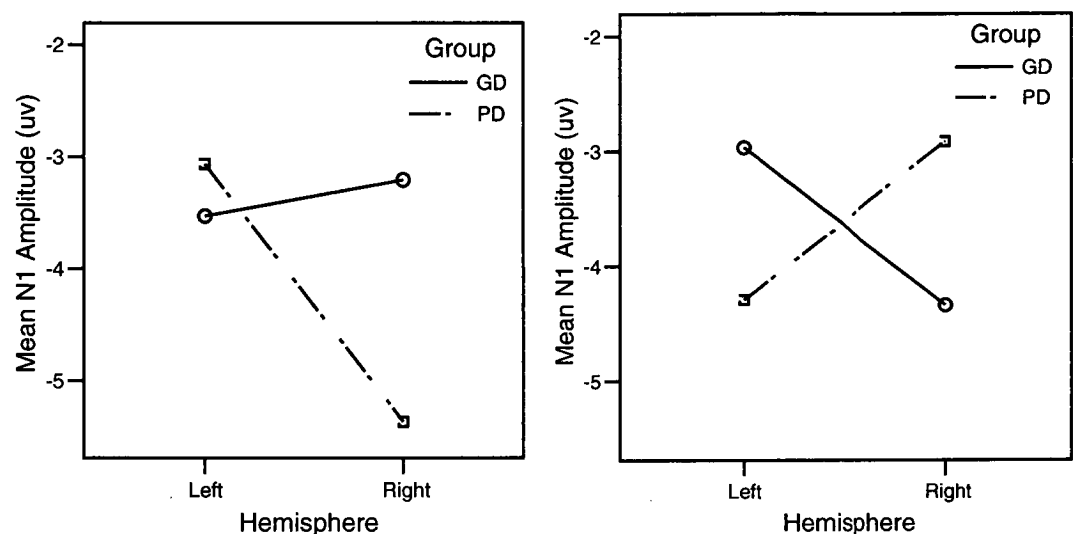


Figure 14. Mean N1 amplitude at left and right hemisphere temporal sites for female and (left) male (right) good decoders (GD) and poor decoders (PD).

N1 Latency

N1 latency was significantly shorter at occipital ($M=155.8$, $SEM=1.73$) and parietal ($M=154.2$, $SEM=1.73$) in comparison to temporal ($M=161.1$, $SEM=1.25$) sites $F(2,60)=14.00$, $MSE=508.92$, $p<.001$, and was significantly shorter at contralateral ($M=150.8$, $SEM=1.51$) in comparison to ipsilateral ($M=163.2$, $SEM=1.76$) sites overall, $F(1,30)=49.38$, $MSE=603.5$, $p<.001$. There was a trend for a VF x Sagittal x Laterality x Group x Sex, interaction $F(2,60)=3.03$, $MSE=94.39$, $p=.057$. The Laterality x VF x Group x Sex interaction was significant at occipital sites, $F(1,30)=5.20$, $MSE=93.94$, $p<.05$, but not at parietal or temporal sites ($ps>.05$). There were no significant Laterality, VF, or Group effects at occipital sites for

females ($ps > .05$). For males there was a significant Laterality x VF x Group interaction at occipital sites, $F(1,11)=8.70$, $MSE=120.5$, $p < .05$ (see Figure 15), such that the VF x Group interaction was significant at ipsilateral, $F(1,11)=15.05$, $MSE=111.0$, $p < .04$, but not at contralateral sites ($p > .05$). Male good decoders tended to show shorter latency for RVF (RH) in comparison to LVF (LH) trials at ipsilateral sites, $F(1,6)=5.73$, $MSE=137.35$, $p = .054$., and male poor decoders showed significantly shorter N1 latency for LVF (LH) in comparison to RVF (RH) trials at ipsilateral sites, $F(1,30)=11.13$, $MSE=79.27$, $p < .05$, and this tended to be shorter in comparison to good decoders, $F(1,11)=3.93$, $MSE=309.2$, $p = .073$.

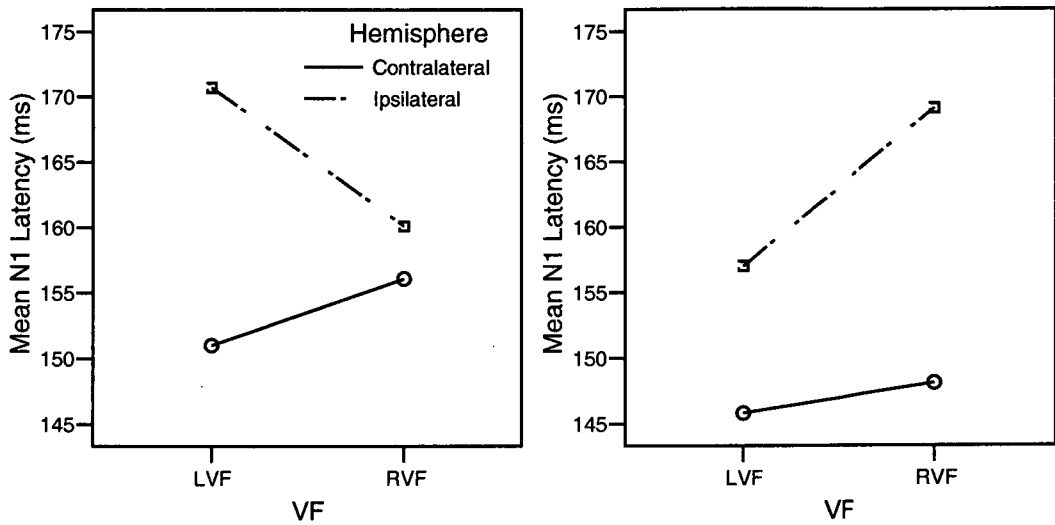


Figure 15. Mean N1 latency for male good decoders (left) and male poor decoders (right) as a function of VF and laterality at occipital sites.

N2 Amplitude

Overall N2 amplitude was significantly greater at occipital ($M=-1.3$, $SEM=0.45$) and temporal ($M=-0.95$, $SEM=0.36$) in comparison to parietal ($M=0.86$, $SEM=0.54$) sites, $F(2,60)=19.13$, $MSE=41.14$, $p < .001$, at contralateral ($M=-0.79$, $SEM=0.43$) in comparison to ipsilateral ($M=-0.15$, $SEM=0.38$) sites, $F(1,30)=3.13$, $MSE=3.13$, $p < .001$, and for the local ($M=-1.1$, $SEM=0.42$) in comparison to the global ($M=0.20$, $SEM=0.41$) task, $F(1,30)=29.69$,

$MSE=23.26$, $p<.001$. The main effect of Group approached significance, such that poor decoders ($M=-1.21$, $SEM=0.58$) tended to show greater N2 amplitude in comparison to good decoders ($M=0.27$, $SEM=0.54$) overall, $F(1,30)=3.44$, $MSE=242.3$, $p=.073$. At temporal sites there was a significant Task x Group interaction, such that the effect of Task was significant for good decoders, $F(1,30)=6.03$, $MSE=209.1$, $p<.05$, but not for poor decoders ($p>.05$), and poor decoders showed significantly greater N2 amplitude in comparison to good decoders for the global task, $F(1,30)=6.56$, $MSE=35.8$, $p<.05$.

There was a significant Sagittal x Hemisphere x Group x Sex interaction, $F(2,60)=4.73$, $MSE=8.17$, $p<.05$, such that the Hemisphere x Group x Sex interaction was significant at temporal sites, $F(1,30)=9.97$, $MSE=18.26$, $p<.01$ (see Figure 16), but not at occipital or parietal sites ($ps>.05$). At LH temporal sites, there was a significant Group x Sex interaction, $F(1,30)=4.21$, $MSE=4.99$, $p<.05$. Female good decoders showed significantly greater N2 amplitude in the LH in comparison to the RH, $F(1,10)=12.56$, $MSE=2.62$, $p<.01$, and female poor decoders tended to show significantly greater N2 amplitude at RH sites in comparison to female good decoders, $F(1,19)=4.58$, $MSE=7.64$, $p=.046$ ($p>.05$ Bonferroni corrected). In contrast, male poor decoders also tended to show greater N2 amplitude at LH in comparison to RH temporal sites, $F(1,5)=5.73$, $MSE=3.22$, $p=.062$, and this tended to be greater in comparison to male good decoders, $F(1,11)=3.71$, $MSE=7.56$, $p=.08$.

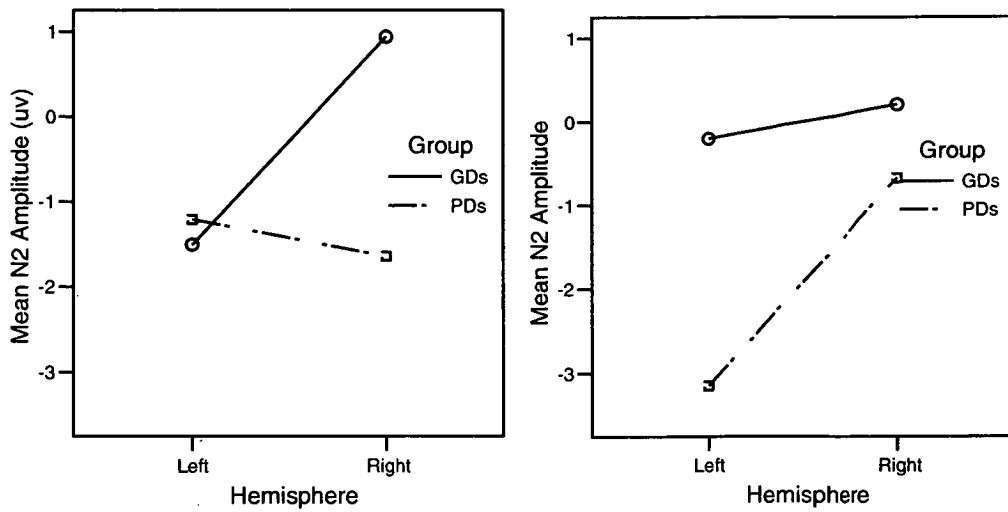


Figure 16. Mean N2 amplitude at temporal sites for female (left) and male (right) poor decoders (PD) and good decoders (GD).

N2 latency

There was a significant main effect of Sagittal site, $F(2,60)=4.86$, $MSE=1449.2$, $p<.05$, such that N2 latency was significantly shorter at temporal ($M=262.5$, $SEM=2.45$) in comparison to occipital sites ($M=269.3$, $SEM=2.67$) and tended to be greater at temporal in comparison to parietal ($M=268.3$, $SEM=3.10$) sites ($p=.062$). N2 latency was also significantly shorter for the global ($M=263.8$, $SEM=2.80$) in comparison to the local ($M=269.6$, $SEM=2.41$) task, $F(1,30)=7.48$, $MSE=1702.3$, $p<.05$, and at contralateral ($M=264.4$, $SEM=2.67$) in comparison to ipsilateral ($M=269.4$, $SEM=2.4$) sites, $F(1,30)=6.23$, $MSE=629.0$, $p<.05$. The main effect of Group was non-significant and there were no significant two-way or three-way interactions involving group ($ps>.05$).

Discussion

Consistent with previous research (Kimchi, 1992; Navon, 1977), there was an overall global precedence effect and an effect of stimulus consistency for the local task in both the RT and accuracy data. Consistent with the findings of Han et al. (2000) there was a RT advantage for target stimuli identified in the RVF relative to the LVF, but this did not interact with any other factor. This RVF advantage could be due to the LH specialisation for the detection of linguistic (letter) stimuli or may be due to the fact that only right hand responses were required in the present paradigm. Poor decoders showed longer RT overall in comparison to good decoders and tended to be less accurate for the global task. This is consistent with previous research finding overall RT differences between dyslexics and age matched controls for both global and local processing tasks (Keen & Lovegrove, 2000), but is not consistent with other research findings indicating a tendency for global rather than local processing in dyslexia (Brannan & Williams, 1987; von Karolyi, 2001; von Karolyi et al., 2003). The accuracy data does provide some support for the hypothesis that an M impairment would selectively impair global processing in poor readers (Keen & Lovegrove, 2000), but this finding should be interpreted with caution as the mean accuracy of both groups was over 95%.

N1 amplitude was maximal at RH parietal sites and for LVF trials consistent with the specialization of the RH for directing spatial attention (Mangun, 1995). N1 amplitude was significantly greater for the local in comparison to the global task at occipital and parietal sites which is consistent with previous ERP studies investigating sustained attention to global and local features (Han et al., 2000). However, this task-related modulation of N1 amplitude was found to be significant for good but not poor phonological decoders. N1 enhancement has been interpreted to reflect the gradient of visual attention (Mangun & Hillyard, 1991) or the demands of visual discrimination within the focus of attention (Vogel & Luck, 2000) and N1 is also modulated by the size of attentional focus such that amplitude is greater when the size of attentional focus is smaller (Luo et al., 2001). Difficulty in expanding and focusing spatial

attention to global and local levels as indexed by the lack of task-related modulation of the N1 component could account for the longer RT observed for poor decoders relative to good decoders regardless of whether global or local features were attended. It is also possible that findings of diffuse allocation of attentional resources in dyslexia (von Karolyi, 2001; von Karolyi et al., 2003; Williams & Bologna, 1985) may be due to a deficit in focussing and widening spatial attention, rather than a propensity towards global processing per se. For example, it has been suggested that global processing requires an expansion of the spotlight/zoom lens, whereas local processing indicates a narrowing of the attentional focus (Stöffer, 1994; Weber et al., 2000).

Both N2 amplitude and latency were also modulated by task, such that greater amplitude and longer latency was observed for the local in comparison to the global task which is consistent with previous research (Han & He, 2003; Han et al., 2000; Heinze et al., 1998). Poor decoders tended to show greater N2 amplitude in comparison to good decoders overall, and did not show the same reduction in N2 amplitude for the global task that was observed among good decoders. The posterior N2 is thought to reflect a non-spatial attentional filtering process involved in isolating one local item from another (Han & He, 2003) and suppressing distracting information (Luck, 1995). Thus the greater N2 amplitude observed for poor decoders may reflect compensatory processing at a later stage due to a deficiency in early spatial selection.

The findings of both neuropsychological (lesion) (Lamb & Robertson, 1988; Lamb, Robertson & Knight, 1989, 1990; Robertson, Lamb & Knight, 1988, cited in Basso & Lowery, 2004) and neuroimaging studies (Fink et al., 1999; Martinez et al., 1997; Weber et al., 2000; Yamaguchi et al., 2000) indicate that local processing of letters is mediated by posterior areas of the LH, whereas global processing is mediated by posterior areas in the RH. These differences are thought to represent a processing bias and it is likely that both hemispheres are able to process global and local but differ in their efficiency (see Hübner & Malinowski, 2002). However, it has also been argued that global/local processing and visual spatial perception share

common underlying neural substrates (Basso & Lowery, 2004) that are both lateralised to the RH. The sustained/selective paradigm and unilateral presentation employed by the present study was not optimal for investigating hemispheric asymmetries in global/local processing (see Han & He, 2003; Heinze et al., 1998).

Although the present study was not necessarily designed to investigate sex differences, males showed a RT advantage in comparison to females for the global task. This is consistent with neuropsychological findings indicating an advantage for males relative to females in both visual spatial performance (Halpern, 1992) and global processing (Basso & Lowery, 2004; Kramer, Ellenberg, Leonard, & Share, 1996). There were also overall differences in the hemispheric lateralisation of ERP components at temporal sites that differed as a function of both sex and decoding group. Female poor decoders and male good decoders showed greater N1 amplitude at RH relative to LH temporal sites and relative to female good decoders and male poor decoders. For N2 amplitude, female good decoders and male poor decoders showed greater N2 amplitude at LH relative to RH temporal sites and relative to female poor decoders and male good decoders. For males there were also trends for lateralisation effects on N1 latency at ipsilateral occipital sites, such that male poor decoders tended to show shorter latency in the LH (LVF) and male good decoders tended to show shorter latency in the RH (RVF).

The greater amplitudes observed in the RH for male good decoders may be related to the global processing advantage observed in the behavioural data. Further, greater activation in the LH for females and the RH for males is consistent with sex differences in hemispheric specialisation (Gur et al., 2000) and may reflect biases in processing visual stimuli. Poor phonological decoders showed a reversal of these hemispheric differences such that female good decoders showed greater amplitudes in the RH and male good decoders showed greater amplitudes in the LH. This pattern of hemispheric differences is also similar to that observed in Experiment 2. However, due to the relatively low number of subjects in each group, and the

marginal levels of statistical significance of some analyses, these effects should be interpreted with caution and further research is required to explore this possibility further.

Previous research findings indicate visual field differences between dyslexics and good readers, characterised by LVF inattention and RVF distractibility or diffuse allocation of attention, suggestive of a RH parietal abnormality (Facoetti & Molteni, 2001). However, studies that have supported this hypothesis have typically included a greater number of male dyslexic and control participants. The findings of the present study indicate a reduction of N1 and N2 ERP amplitudes in the RH for male poor decoders. Thus it is possible that these previous findings are specific to male dyslexics, but further research is required to investigate this hypothesis further.

The present study has shown that poor phonological decoders are slower than good phonological decoders when attention is directed to both the global and local levels of hierarchical stimuli. This was accompanied by a lack of task-related modulation of the posterior N1 component and the N2 component at temporal sites. These findings indicate differences in the allocation of spatial attention and bottom-up perceptual filtering respectively. It is possible that poor decoders made greater use of the later perceptual filtering stage (N2) due to inefficient early spatial selection processes. Whereas there was little evidence of hemispheric asymmetries in global and local processing in the present study, there was evidence for sex differences in the lateralisation of ERP components, such that amplitudes were greater in the RH and LH for male and female good decoders respectively, and a reversal of this asymmetry was observed for poor phonological decoders. These latter findings should be interpreted with caution but indicate a need for further research to examine this hypothesis. Experiment 4 in the current series of experiments aimed to investigate visual field differences in attentional processing and lateralisation of ERP components in greater detail with the use of a covert orienting task.

CHAPTER 9 - EXPERIMENT 4: ELECTROPHYSIOLOGICAL INDICES OF COVERT ORIENTING IN GOOD AND POOR PHONOLOGICAL DECODERS

Covert orienting or the shifting visual attention without overt eye or head movements is most commonly investigated using cueing tasks in which targets are preceded by valid or invalid spatial cues (Posner, 1980; Posner et al., 1980b). Compared to neutral spatial cues, valid and invalid cues produce behavioural (RT and accuracy) benefits and costs respectively. RT benefits are thought to result from perceptual facilitation that is due to either faster intake of information or better perceptual representations (Mangun, 1995). Whereas central or symbolic cues are thought to generate persistent voluntary shifts in attention that are not dependent on cue-to-target SOA, peripheral cues are thought to engage a faster exogenous mechanism that is dependent on the cue-to-target SOA (Müller & Rabbitt, 1989; Posner et al., 1980b; Yamaguchi, Tsuchiya, & Kobayashi, 1994). Peripheral cues produce RT facilitation as early as 50-100ms, but with longer SOAs (300ms or more), inhibition of return may occur such that RT is longer for valid relative to invalid trials (Jonides, 1981; Müller & Rabbitt, 1989; Posner & Cohen, 1984). Inhibition of return disappears at longer SOAs, and is delayed when cues are informative about target locations, suggesting the involvement of a voluntary mechanism (Müller & Rabbitt, 1989) (for a recent review see Klein, 2000)

Dyslexics show an overall increase in RT relative to controls on covert orienting tasks employing both central (Facoetti et al., 2000b; Jonkman et al., 1992; Wijers et al., 2005) and peripheral cues (Facoetti et al., 2003b; Facoetti et al., 2003c; Facoetti et al., 2000b). The findings of some studies indicate greater differences between dyslexics and controls at short rather than long SOAs and for peripheral rather than central cues, suggestive of an automatic orienting deficit (Brannan & Williams, 1987; Facoetti et al., 2000b; Heiervang & Hugdahl, 2003). Other research findings indicate that dyslexics differ from controls in terms of the facilitative effects of spatial attention. For example, Brannan and Williams (1987) found that good but not poor readers show greater accuracy when cues reliably (80%) predicted the location of targets

compared to a task when target prediction was equi-probable (50%). Using a normative sample, Kinsey et al. (2004) found that a brief spatial cue was less effective in drawing attention away from or towards a visual target in poor relative to good readers during a cued coherent motion task. Further, performance on valid trials contributed significant but small amounts of unique variance to the prediction of both nonword (11%) and irregular word reading (8%). Similarly the difference between valid and invalid trials contributed unique variance to the prediction nonword reading accuracy (12%).

Differences have also been observed between dyslexics and controls in terms of inhibitory processing during the performance of covert orienting tasks. In an uninformative peripheral cueing task, dyslexics showed some facilitation at short SOAs coupled with a lack of inhibition of return at longer SOAs (Facoetti et al., 2003b). In a peripheral cueing experiment that included neutral as well as valid and invalid trials, Facoetti et al. (2003c) found that whereas normal readers showed both facilitation (benefits) and inhibition (costs) relative to neutral trials, dyslexic children showed benefits but not costs, suggesting a deficit in the suppression or inhibition of unattended stimuli. Roach and Hogben (2004) investigated spatial cueing with a single fixation search task in five adult dyslexics selected on the basis of nonword reading ability. Cueing the location of a target removed much of the increase in RT associated with increasing numbers of distractors in normal readers but not in dyslexics. These findings are also consistent with other research suggesting that dyslexics have difficulty inhibiting stimuli that are not the current focus of attention (Brosnan et al., 2002; Facoetti & Turatto, 2000; Geiger & Lettvin, 2000).

The findings of some covert orienting studies indicate visual field differences between dyslexics and controls suggestive of underlying differences in cerebral lateralisation or hemispheric control. For example, Brannan and Williams (1987) found that good readers, but not poor readers, showed a significant RVF accuracy advantage in a peripheral cueing experiment, which is consistent with processing differences in the language dominant LH. Using

a cued dot detection task, Facoetti et al. (2001) found that under peripheral cueing conditions (and to a lesser extent under central cuing conditions), dyslexic children showed a spatial cueing effect for LVF but not RVF trials, such that RT was faster for invalid cues in the RVF than LVF. It was suggested that while facilitation was equal in each visual field, the mechanism for contralateral inhibition occurred in the LVF but not the RVF, and therefore suppression of the RVF was absent when the cue was initially presented in the LVF (Facoetti et al., 2001). In a further investigation it was found that right attentional inhibition characterised by decreased RT for RVF invalid trials was present in dyslexic children with impaired nonword reading, but not in dyslexics without impaired nonword reading or controls (Facoetti et al., 2006). Further, dyslexics with a nonword reading deficit also showed longer reaction times to invalid trials in the LVF compared to the other two groups.

The differences observed between dyslexics and controls on covert orienting tasks have often been interpreted to reflect differences in the functioning of the posterior parietal cortex (Facoetti et al., 2000b; Facoetti et al., 2001). Damage to the RH parietal cortex has been shown to result in less inhibition of the opposite hemisphere (Ro et al., 1998), and there is evidence that the RH is involved in directing attention to both visual fields whereas the LH is involved in directing attention to the RVF (Corbetta, Miezin, Shulman, & Peterson, 1993). Other studies employing different attentional paradigms have also reported evidence for LVF inattention in dyslexia which is also consistent with a RH parietal deficit (Eden et al., 2003; Facoetti & Turatto, 2000; Hari et al., 2001).

The time course and functional properties of selective attention can be investigated by comparing posterior ERP waveforms elicited by attended and unattended stimuli. Attention directed towards a specific visual region has been shown to elicit larger P1 and N1 amplitude to stimuli presented in that region, particularly at posterior contralateral sites (for reviews see Eimer, 1998; Hillyard & Anllo-Vento, 1998; Luck, Woodman, & Vogel, 2000; Mangun, 1995). Whereas attentional modulation of P1 and N1 amplitude has typically been observed in the

absence of latency changes, some studies have also reported attentional modulation of P1 and N1 latency such that latency is shorter for valid relative to invalid trials (Anllo-Vento & Hillyard, 1995; Wascher & Tipper, 2004). The attentional modulation of P1 and N1 components has been argued to reflect sensory gain or amplification mechanisms in extra-striate areas which act to decrease signal to noise ratio and facilitate perceptual processing of attended stimuli (see Clark & Hillyard, 1996; Hillyard et al., 1998; Hopf et al., 2002; Mangun, 1995; Mangun et al., 2001). This attentional modulation is thought to act like a spotlight to facilitate processing of information within the bounds of the current attentional focus (Mangun et al., 2001). Attentional modulation of P1 and N1 amplitude is observed for both sustained and trial-by-trial cuing paradigms. However, despite consistent reaction time effects, response relevance of unattended locations, stimulus discriminability, cue type (central, peripheral), cue-to-target SOA, and cue informativeness (the proportion of valid trials) all influence P1 and N1 attention effects, suggesting that modulation is not necessarily obligatory (Doallo et al., 2004, 2005; Eimer, 1994, 1996, 1998). Further, various dissociations have been found between the P1 and N1 attention effects suggesting that they may reflect different underlying mechanisms.

The N1 component consists of three sub-components: the anterior N1 (~140ms), an occipito-parietal component (150-160ms), and an occipito-temporal component (170-200ms) (Clark & Hillyard, 1996; Johannes et al., 1995). Mangun (1995) tentatively argued that occipital-temporal N1 reflects processing within the ventral pathway, whereas the parietal N1 component is related to processing within the dorsal visual pathway. However the findings of some source localisation studies do not necessarily support this hypothesis (e.g., Wijers et al., 1993). Luck et al. (1994) found that posterior N1 amplitude was greater for valid in comparison to neutral cues suggesting that it reflects enhancement of processing at attended locations. N1 attention effects are greater for discrimination tasks (Mangun & Hillyard, 1991) suggestive of a limited capacity process related to the discrimination of stimuli at attended locations (Luck, 1995; Vogel & Luck, 2000). However, it has also been suggested that the N1 attention effect reflects the top-down

modulation of stimulus discrimination processes in occipito-temporal areas of the ventral visual processing stream (Hopf et al., 2002).

The posterior N1 component is consistently modulated by attention at both contralateral and ipsilateral sites during the performance of central cueing tasks and particularly those that require discrimination rather than detection (Doallo et al., 2005; Hillyard, Luck, & Mangun, 1994; Mangun & Hillyard, 1991). Consistent with the time course of endogenous orienting, this modulation is greater for long (700ms) relative to short (200ms) SOAs (Eimer, 2000). In research employing informative peripheral cues with long SOAs, attentional modulation of the N1 component is also typically found (Eimer, 1994; Hillyard et al., 1994), whereas peripheral cueing studies using uninformative cues (Eimer, 1994; McDonald, Ward, & Kiehl, 1999) and short cue-to-target SOAs have revealed inconsistent findings (Doallo et al., 2005; Eimer, 2000; Fu, Fan, Chen, & Zhuo, 2001).

The modulation of the P1 component starts at about 80ms post-stimulus and is thought to index early activity within areas of the ventral visual processing stream (Luck et al., 2000). The findings of both ERP and functional imaging studies suggest a neural generator in ventral lateral occipital areas consistent with an extra-striate source (the fusiform gyrus and surrounding regions) (Clark & Hillyard, 1996; Heinze et al., 1994; Johannes et al., 1995; Martinez et al., 1999). P1 amplitude is often modulated by attention in trial-by-trial central cueing paradigms such that greater amplitude is observed for valid relative to invalid trials (Mangun, 1995) (but see Doallo et al., 2004; Nobre, 2000). However, in contrast to the N1 component, P1 amplitude is reduced for invalid trials compared to neutral trials suggesting that the P1 effect reflects the suppression of information from unattended locations rather than the facilitation of attended locations (Luck et al., 1994).

In research employing peripheral cueing paradigms P1 modulation is dependent on the cue-to-target SOA and cue informativeness. Enhancement of the contralateral P1 component has been found for short SOAs (100-300ms) with both uninformative (Doallo et al., 2004; Hopfinger

& Mangun, 1998, 2001) and informative peripheral cues (Fu et al., 2001) (but see Doallo et al., 2004) suggesting that non-predictive spatial cues summon attention automatically in order to enhance perceptual processing (Hopfinger & Mangun, 1998). However, P1 amplitude is not modulated by peripheral cues at longer SOAs (Anllo-Vento & Hillyard, 1995; Hopfinger & Mangun, 2001). Further, when peripheral cues are uninformative, there is a reversal in the P1 effect such that greater amplitude (posterior ipsilateral) is observed for invalid relative to valid trials (McDonald et al., 1999; Prime & Ward, 2004, 2006; Wascher & Tipper, 2004). Ipsilateral P1 reductions are often accompanied by behavioural inhibition of return effects suggesting perceptual suppression (Wascher & Tipper, 2004) or a perceptual inhibition of return mechanism (Prime & Ward, 2006). Further, when discrimination rather than detection paradigms are used ipsilateral P1 reduction has been observed with no inhibition of return (Eimer, 1994; Hopfinger & Mangun, 1998) and vice versa (Hopfinger & Mangun, 2001).

Whereas several electrophysiological studies have examined covert orienting in relation to reading ability (Harter et al., 1989; Jonkman et al., 1992; Licht et al., 1990; Wijers et al., 2005), none have employed the same paradigm and each differs in terms of the ERP timeframes investigated and the analyses performed on the data making direct comparison difficult. Further, none of these studies have specifically investigated the modulation of the early exogenous components (P1, N1) of the ERP waveform as a function of cue validity. The following studies investigated modulation of early ERP waveforms during trial-by-trial cuing paradigms in which a response was required for targets that were preceded by valid cues (relevant trials), while responses were withheld for stimuli that were preceded by invalid cues (irrelevant trials).

Harter et al. (1989) found no behavioural differences in the performance of reading disabled ($n=12$) and non reading disabled ($n=15$) boys (8-12yrs) diagnosed with or without concurrent ADHD during the performance of an orienting task with uninformative central cues. N1 amplitude (fixed latency 200ms) was greater for relevant in comparison to irrelevant stimuli at occipito-central sites and greater for those with a reading disability in comparison to normal

readers particularly for relevant trials, particularly in the RH for LVF trials. The difference in N1 amplitude between relevant and irrelevant trials correlated significantly with Boder reading level ($r=-0.53$). These findings were interpreted to indicate a reduction in inter-hemispheric competition or inhibition due to a lesion in the LH, or enhanced spatial attention or visuo-spatial strength in peripheral vision, possibly reflecting compensation for reduced later non-spatial target selection as indexed by a reduction in P300 amplitude in the LH.

Jonkman et al. (1992) found that P-type ($n=21$) and L-type ($n=22$) dyslexics were slower to respond and less accurate in comparison to normally reading ($n=22$) Dutch children. Within 100-200ms after the presentation of the target, normal readers showed greater positivity for invalid than valid trials, P-type dyslexics showed greater positivity for valid in comparison to invalid trials (due to a reduction in positivity for invalid trials), and L-type dyslexics showed greater positivity for LVF in comparison to RVF trials overall. The early positivity investigated by Jonkman et al. is likely to correspond to the P1 component and indicates differences in the modulation of this component in different subtypes of dyslexia.

The present study aimed to investigate spatial attention in good and poor phonological decoders using a trial-by-trial covert orienting paradigm in which letter discriminations were required at locations preceded by valid, invalid, and neutral informative cues that were presented either centrally or peripherally. This paradigm differs from the ERP studies outlined above in relation to dyslexia, as a response is required for all stimuli allowing for attentional modulation of both behavioural and ERP responses to be examined in greater detail. The paradigm used in the present study is also more similar to those that have investigated the attentional modulation of early ERP components. Due to the inconsistent behavioural and electrophysiological effects observed in exogenous peripheral cueing paradigms with short SOAs, and the fact that peripheral cueing may involve sensory interactions between the cue and the target stimuli (Mangun, 1995), the present study uses both peripheral and central cues preceding targets by

relatively long SOAs (400ms, 900ms). As such, the present experiment is likely to tap endogenous rather than exogenous covert orienting mechanisms.

Shorter RT and greater accuracy is expected for valid in comparison to invalid trials and benefits and costs are expected when reaction times to valid and invalid trials are compared to neutral trials respectively. If poor phonological decoders differ in terms of attentional facilitation and inhibition, behavioural differences are expected such that less benefits and costs are observed respectively. The N1 and P1 components of the ERP waveform are expected to be modulated by attention such that greater amplitude and shorter latency is observed for valid in comparison to invalid trials. However, for the discrimination tasks used in the present study, the P1 component may be modulated differently, such that greater amplitude is observed for invalid in comparison to valid trials, particularly at ipsilateral sites. Behavioural differences between good and poor decoders are expected to reflect differences in the attentional modulation of the N1 and P1 components. Considering recent findings of asymmetrical visual fields of attention in dyslexia characterised by LVF mini-neglect, and suggestive of a RH parietal deficit (Facoetti & Turatto, 2000; Facoetti et al., 2001; Hari et al., 2001), a further aim of this study was to investigate behavioural differences in each visual field and the associated lateralisation of the P1 and N1 components at parietal sites.

Method

Participants

This study was approved by the University of Tasmania Human Research Ethics Committee and all participants gave written informed consent prior to participation. Thirty-six first year psychology students at the University of Tasmania participated in the experiment as part of their course requirement. All participants had normal or corrected to normal vision and exclusion criteria included a history of drug, alcohol, or tobacco abuse, psychiatric or neurological disorder, head trauma, seizure, and those currently receiving medication. Good (10

female, 9 male) and poor (11 female, 9 male) phonological decoders were selected from a sample of over 300 Psychology 1 students on the basis of Martin and Pratt Nonword Reading Test scores (Martin & Pratt, 2001). One good and two poor phonological decoders were left handed, the remaining participants were right handed as measured by the Edinburgh handedness inventory (Oldfield, 1971). The scores of good phonological decoders ranged from 49-54 (out of a possible score of 54) and the scores of poor phonological decoders ranged from 19-43. Norms for the Nonword reading test are available for samples up to 17 years of age (Martin & Pratt, 2001). The mean score of good decoder group was in the 81st percentile (> 17 years reading age equivalent) and the mean score of poor decoders was in the 18th percentile (10-11 years reading age equivalent) of this norming group.

Ravens Advanced Progressive Matrices (APM) was administered as a measure of non-verbal general intelligence (Raven et al., 1994). Other reading measures included the Word Identification and Comprehension subtests from the Woodcock-Johnson (WJ) Reading Mastery Tests (Woodcock, 1987), The National Adult Reading Test (NART: Nelson & Willison, 1991), an irregular word reading test (see Appendix A), and Reading Accuracy and Reading Rate measures from the Neale Analysis of Reading Ability. Other measures included the Digit Span, Vocabulary, Symbol Coding and Symbol Copy sub-tests from the Weschler Adult Intelligence Scale (WAIS-III: Wechsler, 1997).

Table 5 shows mean age, APM raw score, and scores on reading and cognitive measures for each group. No between group differences were found in terms of age, general nonverbal intelligence (APM), digit span forwards or backwards and symbol copy. Good decoders had significantly higher raw scores in comparison to poor decoders on reading measures including: non-word reading, single word identification, irregular word reading, NART, vocabulary, reading accuracy, and reading rate. There was also a tendency for good decoders to have higher passage comprehension and symbol coding scores. A ceiling effect was observed for non-word reading scores as the upper range for inclusion into the study was much smaller than the lower

range to ensure large differences between groups. A ceiling effect was also observed for Neale reading accuracy as good phonological decoders made very few reading errors relative to poor decoders, who showed significantly greater within group variance.

Table 5.

Mean age and raw scores on reading and cognitive measures for good and poor phonological decoders in Experiment 4.

	Good Decoders		Poor Decoders		Sig.
	(n=19)		(n=20)		
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	
Age	20.07	2.95	19.72	3.21	<i>ns</i>
APM /36	24.26	5.62	22.05	4.01	<i>ns</i>
MP Nonword Reading /54	50.58	1.39	35.45	6.65	***
WJ Word Identification /106	97.79	2.94	88.90	5.28	***
Irregular word reading /87	78.32	5.39	65.40	6.50	***
NART /50	31.68	4.12	19.85	4.73	***
Digit Span Forwards /16	11.16	1.80	10.20	2.07	<i>ns</i>
Digit Span Backwards /14	7.53	1.65	6.85	2.11	<i>ns</i>
WJ Comprehension [#] /68	59.21	2.84	57.30	3.74	<i>p</i> =.082
WAIS Vocabulary [#] /66	51.63	6.86	44.50	5.98	**
WAIS Symbol Coding [#] /60	79.26	8.68	74.40	7.79	<i>p</i> =.073
WAIS Symbol Copy [#] /133	124.84	13.63	117.30	15.75	<i>ns</i>
Neale Reading Accuracy [#] (%)	98.05	2.44	90.75	5.16	***
Neale Reading Rate [#] (words/min)	149.26	17.19	120.24	19.31	***

Note: * = *p*<.05, ** = *p*<.01, *** *p*<.001, [#] Missing data was substituted with the mean for the group for one good decoder and one poor decoder.

Stimuli and Apparatus

Stimuli were presented on an IBM computer and tasks were programmed using the NeuroScan STIM program. Each trial began with a 200ms presentation of an arrow cue. For the central task the cue was presented at fixation and pointed to the left, right, or both visual fields. For the peripheral task, the arrow cue was presented above target positions at the same eccentricity and appeared in either the LVF, RVF or both. Target stimuli were uppercase letters (M or W) presented for 100ms in the left or right visual hemi-field with a randomized cue-to-target SOA of either 400ms or 900ms (or a cue-to-target interval of 200ms or 700ms respectively). Cue and target stimuli were presented white on a black background and subtended 1 x 1 degrees of visual angle at a viewing distance of 70cm. The inner edge of each target letter was presented 6 degrees of visual to the right or left of fixation. The inter-trial interval was 1500ms. Seventy five percent of directionally informative trials were valid and the target was presented to the visual field indicated by the cue. On 25% of trials the target was presented contralateral to the cued location (invalid). Trials preceded by a double arrow cue (neutral) occurred with the same probability as invalid trials. Four blocks of 320 experimental trials were completed, two for each cue type (central peripheral) with response hand (left, right) and letter (M, W) varied between each block.

Electrophysiological Recording

EEG activity was recorded with a NeuroScan system, consisting of a 32-channel Synamps, SCAN 4.1 software, and Quik-cap with Ag/AgCl electrodes interfaced with a NeuroScan STIM 3.1 computer. EEG was recorded from 32 sites, according to the conventional 10-20 system, and all electrodes were referenced to the mastoids. Horizontal electro-oculographic (EOG) activity was recorded bipolarly from electrodes at the outer canthi of both eyes, and vertical EOG was recorded from electrodes above and below the left eye. Electrode impedance was kept below 5 k Ω . EEG activity was amplified with a bandpass of 0.15-100 Hz

and sampled continuously at a rate of 1000 Hz. EEG data were merged with behavioural files and ocular artefact reduction was conducted by regression and artifact averaging (Semlitsch, Anderer, Schuster, and Presslich, 1986). Data files were epoched offline for a 1000ms epoch commencing 100ms before stimulus onset and baseline corrected to the pre-stimulus interval. High and low voltage cut-offs for artefact rejection were set at 100 μ V and -100 μ V respectively. Correct responses were averaged across letter (M, W) and band-pass filtered (0.5-30 Hz). Posterior P1 and N1 components were determined from grand averaged means as the maximum voltage within the following time frames after target onset: (P1: 90-150ms; N1: 150-230ms).

Procedure

The reading and neuropsychological measures were administered in a screening session of approximately one hour on a day prior to the experimental session. Following set-up for EEG recording, participants were seated in front of a computer monitor, at a viewing distance of 70cm. Four letter discrimination tasks were completed in counterbalanced order. Participants were required to indicate whether each letter presented was an M or W using a two-button response pad. Participants were instructed to respond as quickly and as accurately as possible to all target stimuli irrespective of cue validity and to avoid blinking or overtly moving their head or eyes. Participants took short breaks between tasks to prevent fatigue. The experimental session lasted approximately two hours (including set up for electrophysiological recording) with most participants completing several different experiments within the session.

Design and Data Analysis

Mean RT, accuracy, and mean amplitude and latency of P1 and N1 components were averaged across letter and response hand condition and the effects of experimental manipulations investigated using 2(Task: central, peripheral) x 2(SOA: 200ms, 500ms) x 3(Cue: valid, invalid) x 2(VF: LVF, RVF) repeated measures ANOVAs with two additional between subjects factors of 2(Group: good decoder, poor decoder) x 2(Sex: male, female). At this stage, neutral trials were not used in analyses due to the difficulty in ascertaining the effects of these cues on the allocation of spatial attention, particularly between the two tasks. However, to further investigate the costs (neutral minus valid trials) and benefits (neutral minus valid trials) of spatial attention on reaction time, transformed variables were analysed using 2(Group: good decoder, poor decoder) x 2(Sex: male, female) x 2(Task: central, peripheral) x 2(SOA: 200ms, 500ms) x 2(VF: LVF, RVF) ANOVAs. The effects of experimental manipulations on mean P1 and N1 amplitude and latency at parietal sites were analysed using 2(Group: good decoder, poor decoder) x 2(Sex: male, female) x 3(Task: central, peripheral) x 2(SOA: 400ms, 900ms) x 2(Cue: valid, invalid) x 2(VF: LVF, RVF) x 3 (Sagittal site: occipital, parietal, temporal) x 2 (Laterality: contralateral, ipsilateral) repeated measures ANOVAs. Greenhouse-Geisser corrections were applied where appropriate to control for violations of sphericity. Significant interactions were analysed using break-down ANOVAs with Bonferroni adjusted p-values for analysis of simple effects. Due to the large number of significant interactions in the electrophysiological data, significant interactions involving SOA were not reported because they were not relevant to the specific aims of the study. Only interactions that involved the variables of Cue or VF in combination with Group and/or Sex were analysed further.

Results

Mean Reaction Time

There was a trend for an overall main effect of Group, $F(1,35)=3.81$, $MSE=.042$, $p=.059$, such that RT tended to be shorter for good decoders ($M=.494$, $SEM=.012$) in comparison to poor decoders ($M=.526$, $SEM=.012$). The main effect of Cue was significant, $F(1,35)=64.86$, $MSE=0.0007$, $p<.001$, indicating significantly shorter mean RT for valid ($M=.501$, $SEM=.008$) in comparison to invalid ($M=.518$, $SEM=.008$) trials. There was a trend for an overall Cue x Group interaction, $F(1,35)=3.52$, $MSE=0.0007$, $p=.069$. The effect of Cue was significant for both good decoders, $F(1,17)=37.36$, $MSE=0.0009$, $p<.001$, and poor decoders, $F(1,18)=26.91$, $MSE=0.0005$, $p<.001$, and the main effect of Group approached significance for valid trials, $F(1,35)=4.67$, $MSE=0.022$, $p=.038$ ($p>.05$ Bonferroni corrected) and invalid trials, $F(1,35)=2.91$, $MSE=0.021$, $p=.097$. There was also a significant Cue x Sex interaction, $F(1,35)=7.72$, $MSE=0.0007$, $p<.01$. Breakdown analyses of this interaction indicated that the effect of Cue was significant for both females, $F(1,19)=64.59$, $MSE=0.0007$, $p<.001$, and males, $F(1,16)=12.68$, $MSE=0.0007$, $p<.01$, however the main effect of Sex was not significant for either valid or invalid trials ($ps>.05$). These findings indicate that the effect of Cue was greater for females relative to males.

Although the Cue x Sex x Group interaction shown in Figure 18 was non-significant, $F(1,35)=1.39$, $MSE=0.0007$, $p>.05$, planned comparisons were conducted to investigate the main effect of Cue for male and female good decoders and poor decoders. The main effect of Cue was significant for both female good decoders, $F(1,9)=37.7$, $MSE=0.001$, $p<.001$, and female poor decoders, $F(1,10)=27.9$, $MSE=0.001$, $p<.001$. However, the Cue x Group interaction approached significance for males, $F(1,16)=4.26$, $MSE=0.0007$, $p=.056$, such that the main effect of Cue was significant for male good decoders, $F(1,8)=9.39$, $MSE=0.001$, $p<.05$, and only approached significance for male poor decoders, $F(1,8)=3.55$, $MSE=0.002$, $p=.096$.

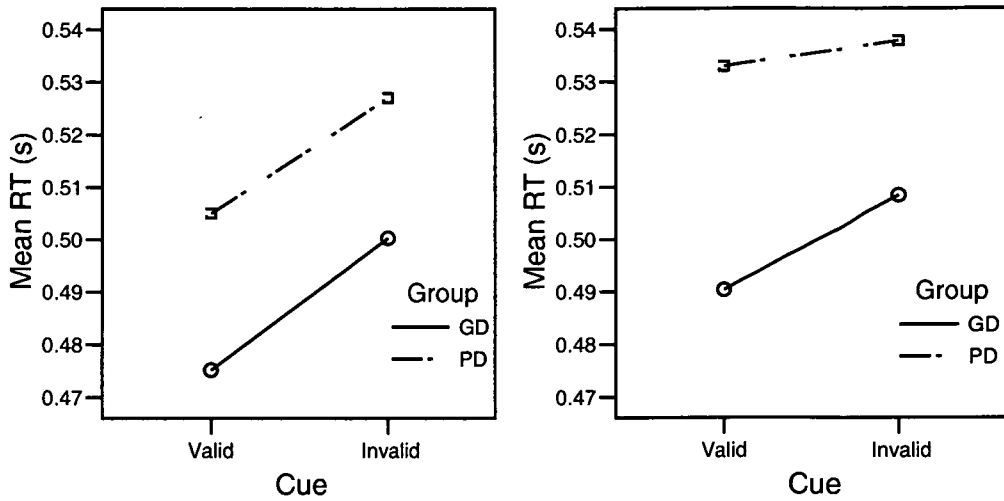


Figure 18. Mean RT on valid and invalid trials for female (left) and male (right) good decoders (GD) and poor decoders (PD).

To investigate visual field differences, separate analyses were performed for the LVF and RVF. The Cue x Group interaction approached significance for LVF trials, $F(1,35)=3.72$, $MSE=0.01$, $p=.062$, such that the effect of Cue was significant for both good decoders and poor decoders ($p<.05$), but the effect of Group approached significance for valid trials $F(1,37)=3.37$, $MSE=0.003$, $p=.074$, indicating longer RT for poor decoders in comparison to good decoders. For RVF trials, there was an overall effect of Group, $F(1,35)=5.27$, $MSE=0.02$, $p<.05$, indicating longer RT for poor decoders in comparison to good decoders overall. When this was analysed separately for each sex, males showed a significant Cue x Group interaction for LVF trials, $F(1,16)=6.17$, $MSE=0.0005$, $p<.05$, such that the effect of Cue was significant for male good decoders, $F(1,8)=13.37$, $MSE=0.001$, $p<.001$, but not male poor decoders ($p>.05$), though there were no significant between group differences ($ps>.05$). Females showed an overall effect of cue for both visual fields ($ps<.05$).

To investigate any differences in central and peripheral cueing manipulations, separate analyses were performed for each task. For the central cueing condition, there were significant main effects of Group, $F(1,35)=4.32$, $MSE=4.32$, $p<.05$, and Cue, $F(1,35)=29.42$, $MSE=0.0008$,

$p < .001$. For the peripheral task, there was a significant main effect of Cue, $F(1,35)=56.88$, $MSE=0.0004$, $p < .001$, and the main effect of Group approached significance, $F(1,35)=3.05$, $MSE=0.022$, $p=.089$. There was also a significant main effect of VF for the peripheral task, $F(1,35)=8.62$, $MSE=.0005$, $p < .01$, indicating shorter RT for RVF in comparison to LVF trials. However, this was modified by a trend for a Group x VF interaction, $F(1,35)=3.16$, $MSE=.0005$, $p=.084$, such that the effect of VF approached significance for good decoders, $F(1,17)=4.67$, $p=.045$ ($p > .05$, Bonferroni corrected), but was non-significant for poor decoders ($p > .05$). Poor decoders tended to show longer RT for RVF trials in comparison to good decoders, $F(1,35)=4.54$, $MSE=.011$, $p=.048$ ($p > .05$, Bonferroni corrected).

Reaction Time Costs and Benefits

For RT benefits (neutral minus valid trials), the main effect of Group approached significance, $F(1,35)=3.19$, $MSE=0.001$, $p=.083$, and the Sex x Group interaction also approached significance, $F(1,35)=3.50$, $MSE=0.001$, $p=.070$, such that the effect of Group was significant for males, $F(1,35)=6.35$, $MSE=0.001$, $p < .05$, but not for females ($p > .05$), indicating greater benefits for male good decoders in comparison to male poor decoders. There was a significant Task x Group interaction for RT benefits, $F(1,35)=4.69$, $MSE=0.0005$, $p < .05$. Poor decoders showed less RT benefits for the central relative to the peripheral task, $F(1,18)=10.43$, $MSE=0.001$, $p < .01$, and in comparison to good decoders, $F(1,35)=7.12$, $MSE=0.001$, $p < .05$.

For RT costs (neutral minus invalid), there was significant Task x VF x Group interaction, $F(1,35)=5.58$, $MSE=0.01$, $p < .05$ (see Figure 19). The Task x Group interaction approached significance for LVF trials, $F(1,35)=3.53$, $MSE=0.001$, $p=.068$, such that poor decoders tended to show greater costs for the central relative to the peripheral task, $F(1,18)=3.38$, $MSE=0.001$, $p=.083$. However, there were no overall effects of Group for either task ($ps > .05$) and no significant effects of Task or Group in the RVF ($ps > .05$).

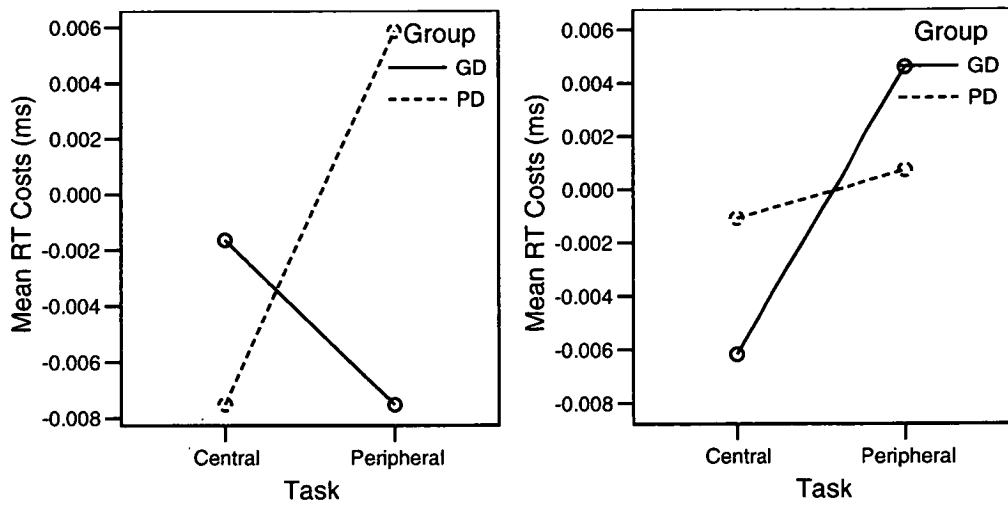


Figure 19. Mean RT costs of good decoders (GD) and poor decoders (PD) as a function of Task for LVF (left) and RVF (right) trials.

Mean Accuracy

There were no significant main effects of any variable on the mean percentage of correct trials ($p > .05$). There was a significant Task x Sex x Group interaction, $F(1,35)=4.75$, $MSE=80.22$, $p < .05$, such that the Sex x Group interaction was significant for the central task, $F(1,35)=4.31$, $MSE=420.72$, $p < .05$. Male poor decoders ($M=92.0\%$, $SEM=2.4\%$) tended to be more accurate than male good decoders ($M=84.8\%$, $SEM=2.4\%$), $F(1,16)=4.80$, $MSE=398.97$, $p=.044$ ($p > .05$, Bonferroni corrected), and female poor decoders ($M=85.4\%$, $SEM=1.9\%$), $F(1,18)=5.58$, $MSE=317.22$, $p=.03$ ($p > .05$, Bonferroni corrected). There was also a significant VF x Cue x Sex x Group interaction, $F(1,35)=4.64$, $MSE=19.10$, $p < .05$, indicating a significant Cue x Sex x Group interaction for RVF trials, $F(1,35)=5.42$, $MSE=21.58$, $p < .05$. The Cue x Group interaction was significant for males, $F(1,16)=7.96$, $MSE=22.46$, $p < .05$, such that male good decoders tended to less accurate on invalid relative to valid trials, $F(1,8)=7.40$, $MSE=22.88$, $p=.026$ ($p > .05$, Bonferroni corrected) and this was significantly lower in comparison to male poor decoders, $F(1,35)=8.12$, $MSE=163.6$, $p < .05$. There were no significant cue effects for male poor decoders and females ($p > .05$).

Electrophysiological analyses

Figures 20 - 23 show grand mean averaged ERP waveforms for male and female good decoders and poor decoders as a function of Cue, Task, VF and Sex. For LVF trials for both the central (Figure 20) and peripheral (Figure 22) tasks, both female good and poor decoders and male good decoders show greater N1 amplitude for valid relative to invalid trials at RH parietal sites (P4). In contrast, for male poor decoders, there appears to be little difference in N1 amplitude between valid and invalid trials. For RVF trials (Figures 21 and 23) male good decoders show greater N1 amplitude at RH parietal sites relative to male poor decoders. In contrast, female poor decoders showed greater N1 amplitude at RH sites relative to female good decoders, and this differs as a function of Cue and Task. Good decoders also show greater N1 amplitude at LH temporal sites (P7) relative to poor decoders and the magnitude of this difference varies as a function of Cue, VF and Sex. Male good decoders seem to show larger P1 amplitude relative to male poor decoders, particularly at parietal sites for the central task (Figures 20 and 21). Relative to the central task, there is a reversal of the attentional modulation of the P1 component for the peripheral task (Figures 22 and 23) such that amplitude is greater for invalid relative to valid trials, particularly at ipsilateral sites (the LH for LVF trials and the RH for RVF trials). Differences in the attentional modulation and lateralisation of the P1 and N1 components as a function of Sex and Group are examined statistically below.

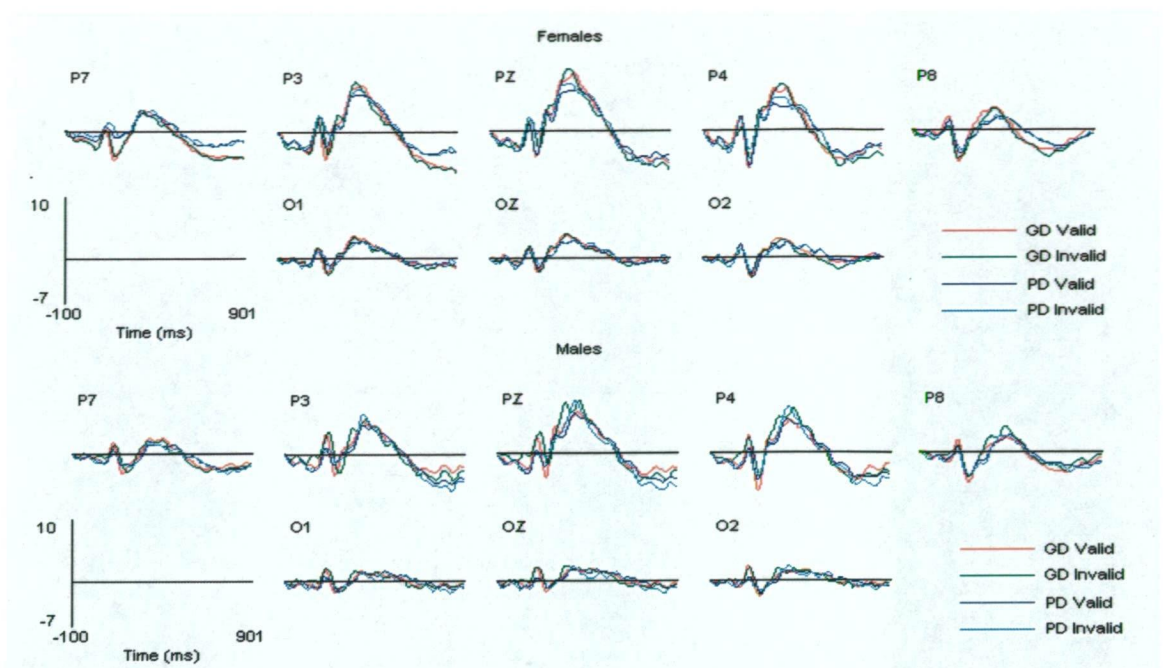


Figure 20. Grand mean averages at posterior sites for LVF centrally cued trials as a function of cue validity and group for females (above) and males (below).

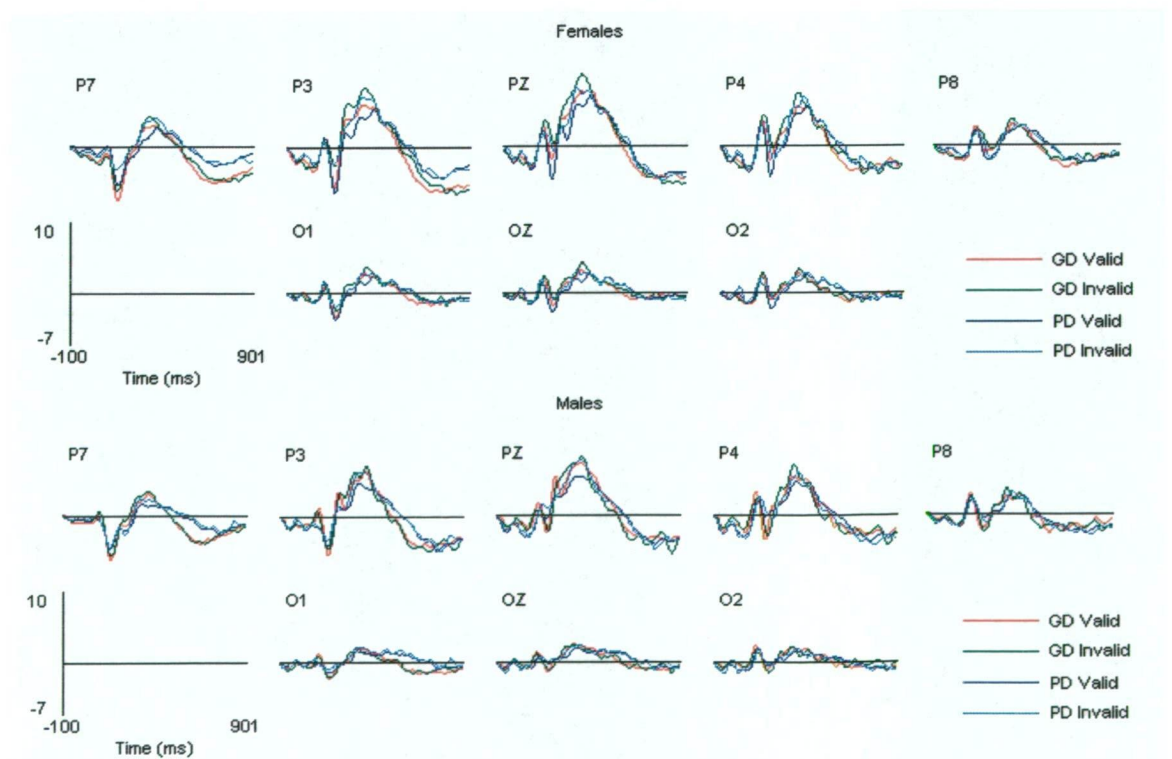


Figure 21. Grand mean averages at posterior sites for RVF centrally cued trials as a function of cue validity and group for females (above) and males (below).

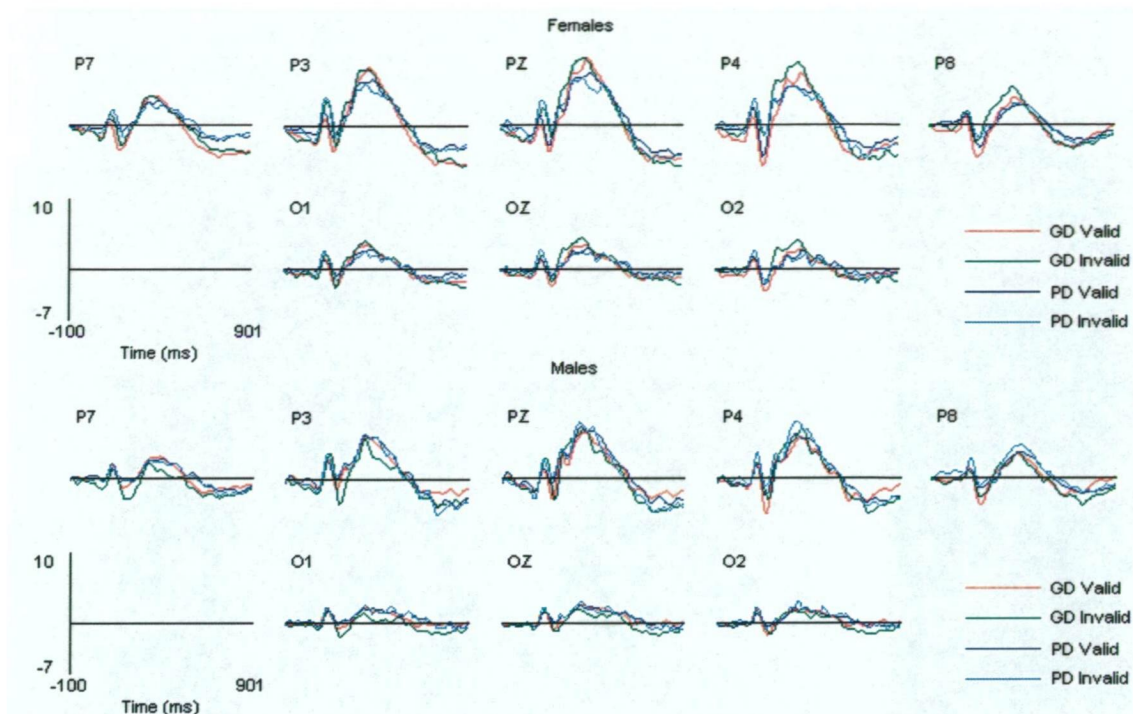


Figure 22. Grand mean averages at posterior sites for LVF peripherally cued trials as a function of cue validity and group for females (above) and males (below).

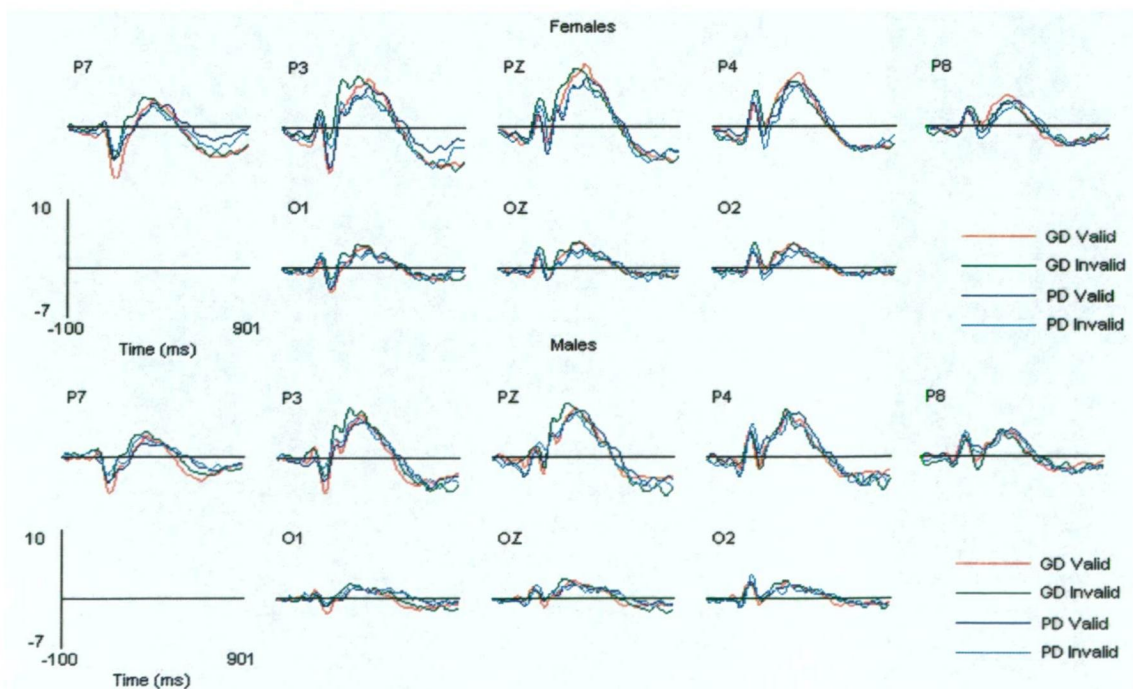


Figure 23. Grand mean averages at posterior sites for RVF peripherally cued trials as a function of cue validity and group for females (above) and males (below).

N1 Amplitude

There was a significant main effect of Sagittal site, $F(1,35)=24.01$, $MSE=42.26$, $p<.001$, indicating significantly greater N1 amplitude at parietal ($M=-4.32$, $SEM=.354$), followed by temporal ($M=-3.72$, $SEM=.246$), and occipital sites ($M=-2.63$, $SEM=.238$) respectively. N1 amplitude was significantly greater at contralateral ($M=-4.32$, $SEM=.304$) in comparison to ipsilateral ($M=-2.78$, $SEM=.237$) sites, $F(1,35)=42.00$, $MSE=50.72$, $p<.001$, for the central ($M=-4.03$, $SEM=.271$) in comparison to the peripheral task ($M=-3.08$, $SEM=.245$), $F(1,35)=35.17$, $MSE=35.17$, $p<.001$, and for the 400ms ($M=-4.90$, $SEM=.315$) in comparison to the 900ms SOA ($M=-2.21$, $SEM=.277$), $F(1,35)=65.7$, $MSE=99.40$, $p<.001$. The main effects of Cue, Group, and VF were non-significant ($ps>.05$). There was a significant Task x Cue x Laterality interaction, $F(1,35)=13.18$, $MSE=6.53$, $p<.01$, the Cue x Laterality interaction was significant for both tasks. N1 amplitude was significantly greater for valid in comparison to invalid trials at contralateral sites for both the central, $F(1,34)=5.31$, $MSE=0.484$, $p<.05$ and peripheral, $F(1,35)=16.27$, $MSE=0.919$, $p<.001$, tasks. However, for the peripheral task N1 amplitude was significantly greater for invalid in comparison to valid trials at ipsilateral sites, $F(1,35)=11.87$, $MSE=1.22$, $p<.01$.

Figure 24 shows a trend for a VF x Laterality x Group interaction, $F(1,35)=3.99$, $MSE=37.33$, $p=.054$. There was a significant VF x Group interaction at ipsilateral sites, $F(1,35)=9.83$, $MSE=6.03$, $p<.01$, such that good decoders showed significantly greater N1 amplitude for LVF in comparison to RVF trials, $F(1,17)=16.4$, $MSE=5.33$, $p<.01$, and this was significantly greater in comparison to poor decoders, $F(1, 37)=6.58$, $MSE=14.69$, $p<.05$. The effect of VF was non-significant for poor decoders ($p>.05$). However, this was qualified by a significant VF x Sex x Group at ipsilateral sites, $F(1,35)=5.12$, $MSE=6.03$, $p<.05$. The VF x Group interaction was significant for females, $F(1, 19)=16.05$, $MSE=2.04$, $p<.01$, but not for males ($p>.05$), such that the effect of VF was significant for female good decoders, $F(1, 9)=28.42$, $MSE=4.22$, $p<.001$, but not for female poor decoders ($ps>.05$). However, there was

also a significant Sex x Group interaction in the RH for RVF trials, $F(1,34)=6.57$, $MSE=16.55$, $p<.05$, such that female poor decoders tended to show greater N1 amplitude at ipsilateral sites for RVF (RH) trials in comparison to female good decoders, $F(1, 19)=5.60$, $MSE=2.20$, $p=.029$ ($p>.05$ Bonferroni corrected).

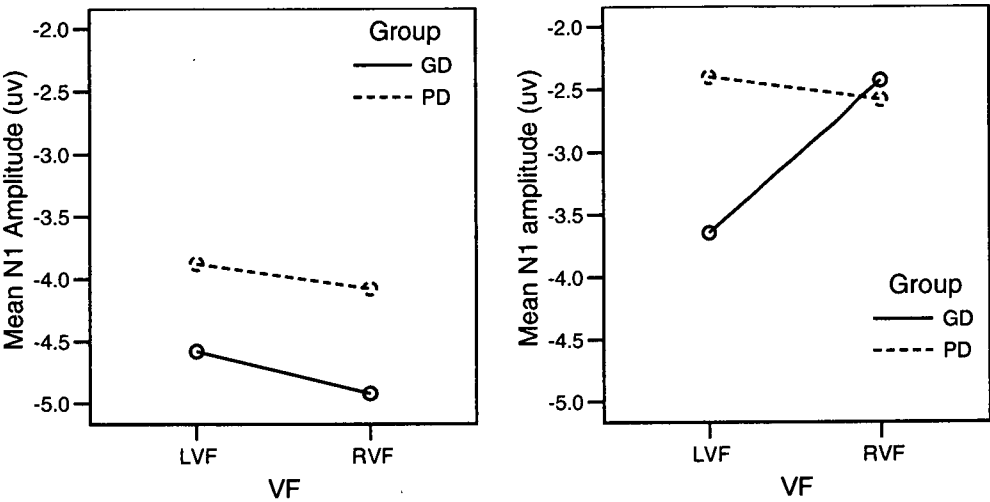


Figure 24. Mean N1 amplitude as a function of VF at contralateral (left) and ipsilateral (right) sites for good decoders (GD) and poor decoders (PD).

There was a significant Sagittal x Laterality x VF x Cue x Group x Sex interaction, $F(1,34)=4.77$, $MSE= p=.015$. To investigate these interactions further separate analyses were conducted at each sagittal site. At temporal sites, there was a significant main effect of Group, $F(1,34)=4.80$, $MSE=18.07$, $p=.035$, such that N1 amplitude was significantly greater for GDs ($M=-4.25$, $SEM=0.356$) in comparison to PDs overall ($M=-3.18$, $SEM=0.338$). There was also a significant Lat x Cue x Sex x Group interactions for LVF trials, $F(1,34)=4.30$, $MSE=1.20$, $p<.05$. At ipsilateral temporal sites when targets were presented in the LVF (LH), the Cue x Sex x Group interaction was significant (see Figure 25), $F(1,34)=7.38$, $MSE=1.08$ $p<.05$. Female GDs tended to show greater N1 amplitude for valid relative to invalid trials, $F(1,9)=5.01$, $MSE=.370$, $p=.052$, and N1 amplitude for valid trials was significantly greater in comparison to female PDs,

$F(1,19)=10.07$, $MSE=2.78$, $p<.01$. In contrast to females, male good decoders showed greater N1 amplitude for invalid in comparison to valid trials, $F(1,7)=7.18$, $MSE=1.51$, $p<.05$, and the effect of Cue was non-significant for male PDs ($p>.05$).

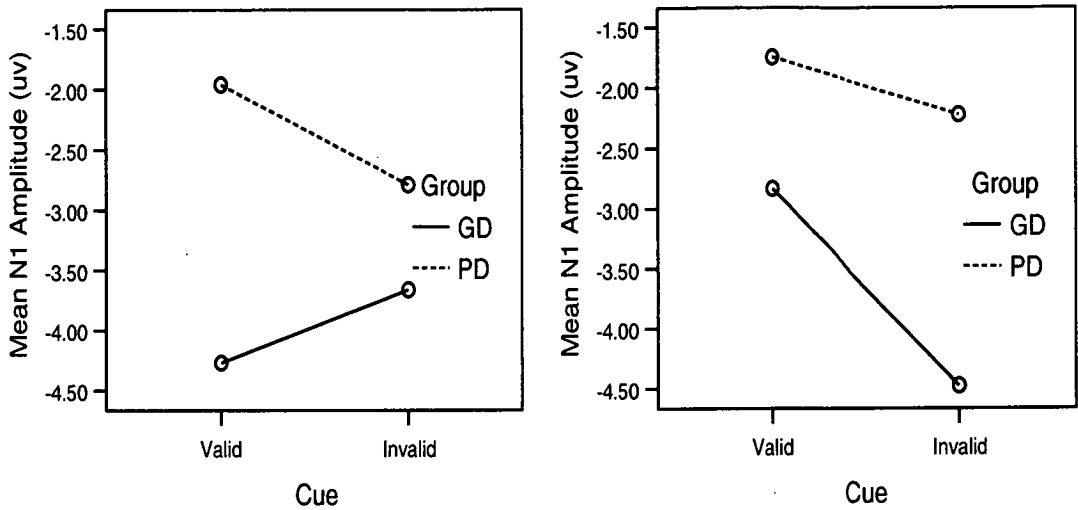


Figure 25. Mean N1 amplitude for LVF trials at ipsilateral (LH) temporal sites for female (left) and male (right) good (GD) and poor phonological (PD) decoders.

At parietal sites there was a significant Laterality x VF x Cue x Group x Sex interaction, $F(1,34)=10.07$, $MSE=4.36$, $p<.01$. However, based on the different effects of Laterality and Cue for each task, separate analyses were conducted at contralateral sites for both tasks and ipsilateral sites for the peripheral task. For the central task, there was a significant VF x Cue x Laterality x Sex x Group interaction at parietal sites, $F(1,35)=9.54$, $MSE=3.00$, $p<.01$. At contralateral parietal sites (see Figures 26-27), the overall effect of Cue was significant for both LVF (RH), $F(1,35)=8.23$, $MSE=2.10$, $p<.01$, and RVF (LH) trials, $F(1,35)=14.75$, $MSE=6.38$, $p<.001$. However, for RVF trials (Figure 26), the Cue x Sex interaction was significant, $F(1,35)=6.47$, $MSE=2.10$, $p<.05$, such that the effect of Cue was significant for females, $F(1,19)=21.30$, $MSE=6.72$, $p<.001$, but not for males ($p>.05$). For LVF (RH) trials (Figure 27),

the Cue x Sex x Group interaction was significant, $F(1,35)=6.47$, $MSE=2.10$, $p<.05$. The overall effect of Cue was significant for females, $F(1,19)=4.63$, $MSE=3.08$, $p<.05$. However, males showed a significant Cue x Group interaction, $F(1,16)=7.02$, $MSE=0.940$, $p<.05$, such that the effect of Cue was significant for male good decoders, $F(1,8)=12.73$, $MSE=0.883$, $p<.01$, but not for poor decoders ($p>.05$). There were no significant differences between good and poor decoders for either sex ($ps>.05$).

For the peripheral task the Laterality x VF x Cue x Sex x Group interaction was also significant at parietal sites, $F(1,35)=4.36$, $MSE=3.70$, $p<.05$. Figures 28-29 show N1 amplitude as a function of VF, Cue, Sex and Group at contralateral sites. N1 amplitude was greater for valid relative to invalid trials at contralateral sites, $F(1,35)=29.37$, $MSE=4.13$, $p<.001$. However, this was qualified by a significant Cue x Sex interaction, $F(1,35)=9.96$, $MSE=4.13$, $p<.05$, such that the effect of Cue was significant for females, $F(1,19)=59.4$, $MSE=2.77$, $p<.001$, but not for males ($p>.05$). For RVF (LH) trials (Figure 28), there was a trend for a Cue x Group interaction for females ($p=.066$). Although the effect of Cue was significant for both female groups ($ps<.05$), it was greater for good decoders in comparison to poor decoders. Males showed an overall effect of Cue for RVF trials ($p=.027$), however, this only approached significance for male good decoders ($p=.075$) and was non-significant for male poor decoders ($p>.05$). For LVF (RH) trials (Figure 29), female good decoders and poor decoders showed a significant effect of Cue ($p<.05$), but the effect of Cue was non-significant for both male good decoders and poor decoders ($ps>.05$).

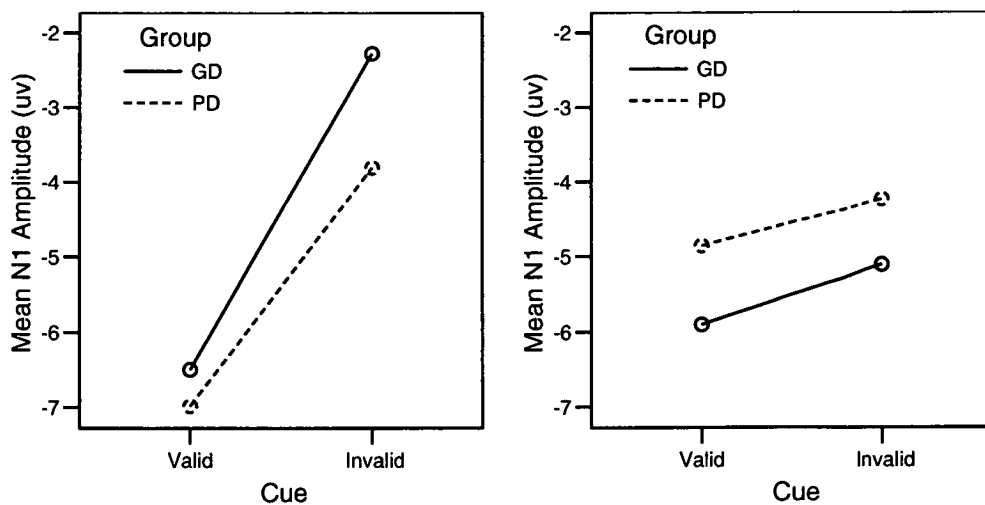


Figure 26. Mean N1 amplitude for RVF trials at contralateral (LH) parietal sites for the central task as a function cue for female (left) and male (right) good decoders (GD) and poor decoders (PD).

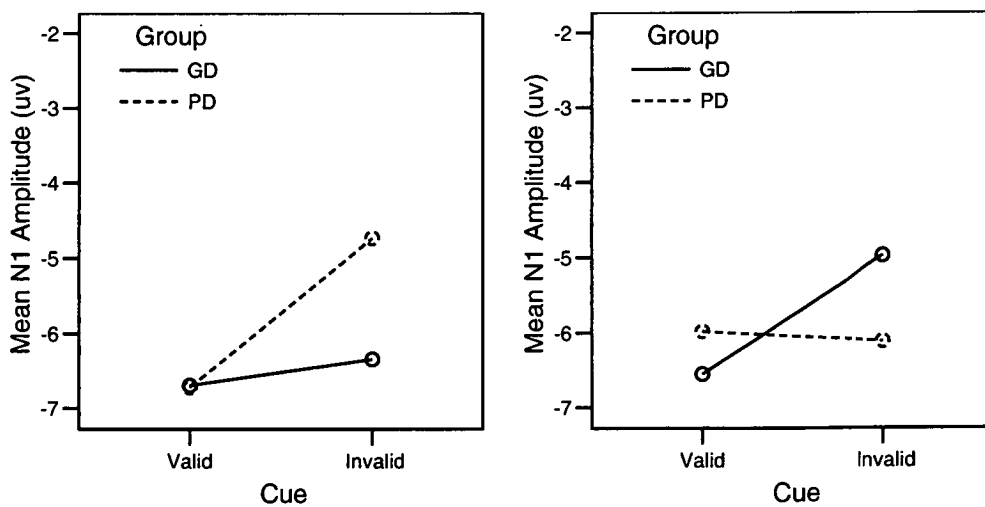


Figure 27. Mean N1 amplitude for LVF trials at contralateral (RH) parietal sites for the central task as a function of cue for female (left) and male (right) good decoders (GD) and poor decoders (PD).

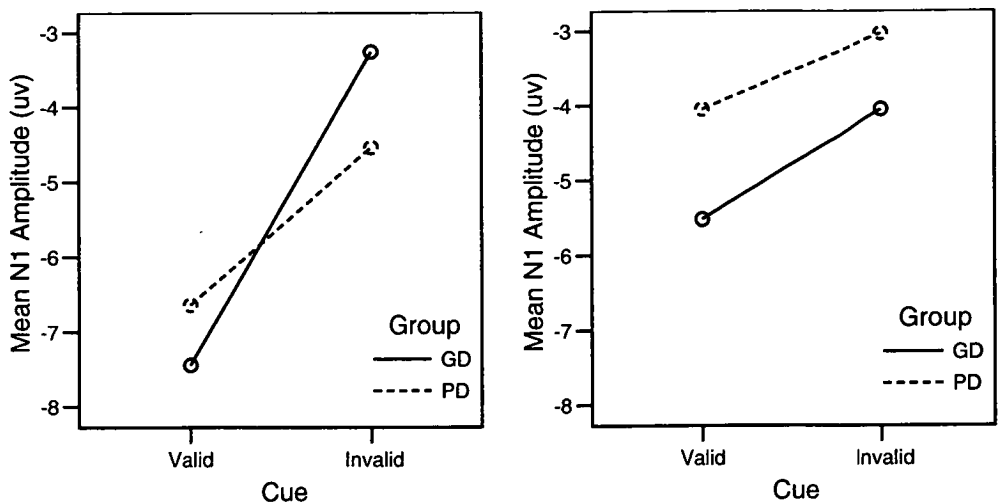


Figure 28. Mean N1 amplitude for RVF trials at contralateral (LH) parietal sites for the central task as a function cue for female (left) and male (right) good decoders (GD) and poor decoders (PD).

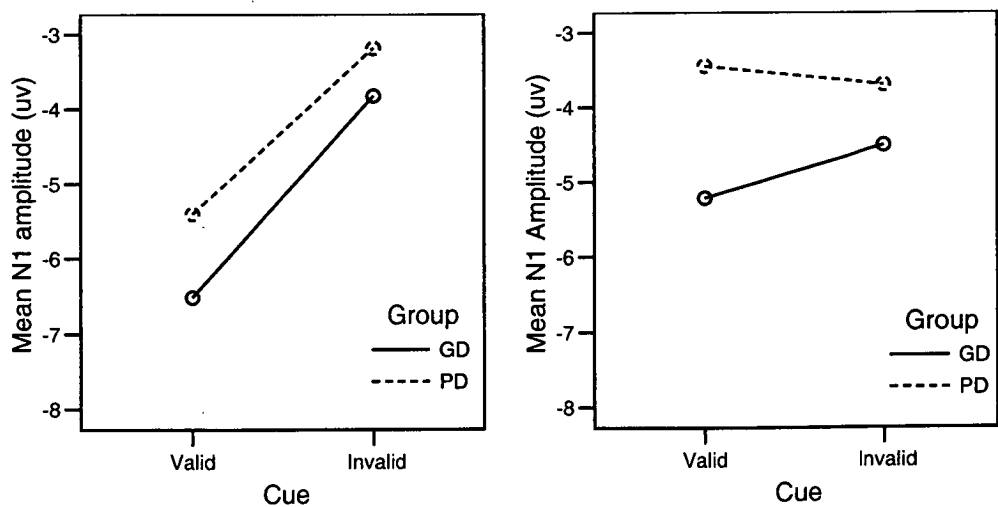


Figure 29. Mean N1 amplitude for LVF trials at contralateral (RH) parietal sites for the central task as a function of cue for female (left) and male (right) good decoders (GD) and poor decoders (PD).

At ipsilateral parietal sites under peripheral cueing conditions, N1 amplitude was significantly greater for invalid than valid trials overall, $F(1,35)=8.71$, $MSE=4.67$, $p<.01$. There was a significant VF x Group interaction at ipsilateral sites, such that good decoders showed greater N1 amplitude for LVF (LH) than RVF (RH) trials, $F(1,35)=11.60$, $MSE=4.64$, $p<.05$, and this was significantly greater in comparison to poor decoders, $F(1,35)=7.34$, $MSE=9.26$, $p<.05$. For LVF (LH) trials, the Cue x Group x Sex interaction was significant (see Figure 30), $F(1,35)=5.07$, $MSE=3.63$, $p<.05$. Male good decoders showed significantly greater N1 amplitude for invalid relative to valid trials, $F(1,35)=12.39$, $MSE=2.77$, $p<.01$. The effect of Cue approached significance for male poor decoders ($p=.091$), and was non-significant for female good decoders and poor decoders ($ps>.05$). Male good decoders showed significantly greater N1 amplitude in comparison to male poor decoders overall, $F(1,16)=7.37$, $MSE=11.00$, $p<.05$. There was a significant Sex x Group interaction for RVF (RH) trials (see Figure 31), $F(1,35)=6.37$, $MSE=13.70$, $p<.05$, such that female poor decoders showed greater N1 amplitude in comparison to female good decoders, $F(1,19)=6.08$, $MSE=3.70$, $p<.05$.

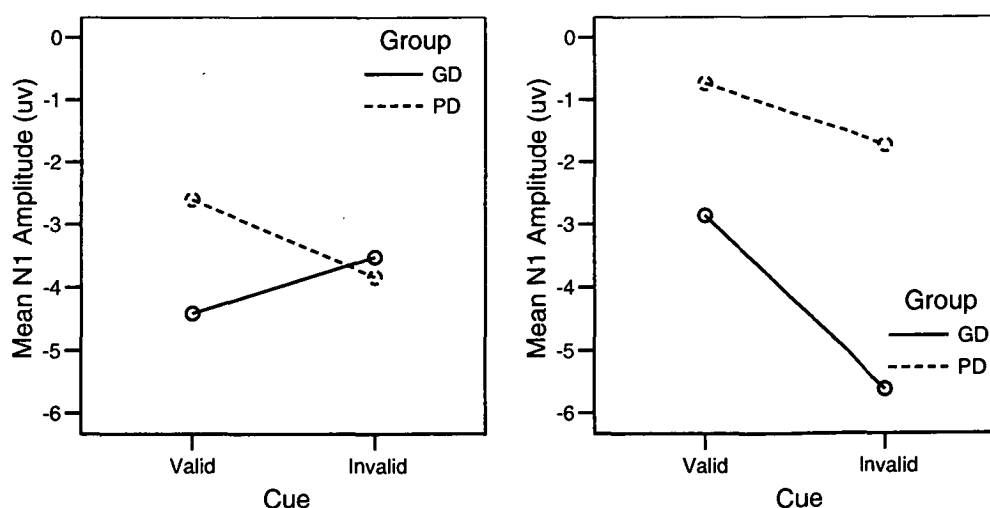


Figure 30. Mean N1 amplitude for LVF trials at ipsilateral (LH) parietal sites for the central task as a function cue for female (left) and male (right) good decoders (GD) and poor decoders (PD).

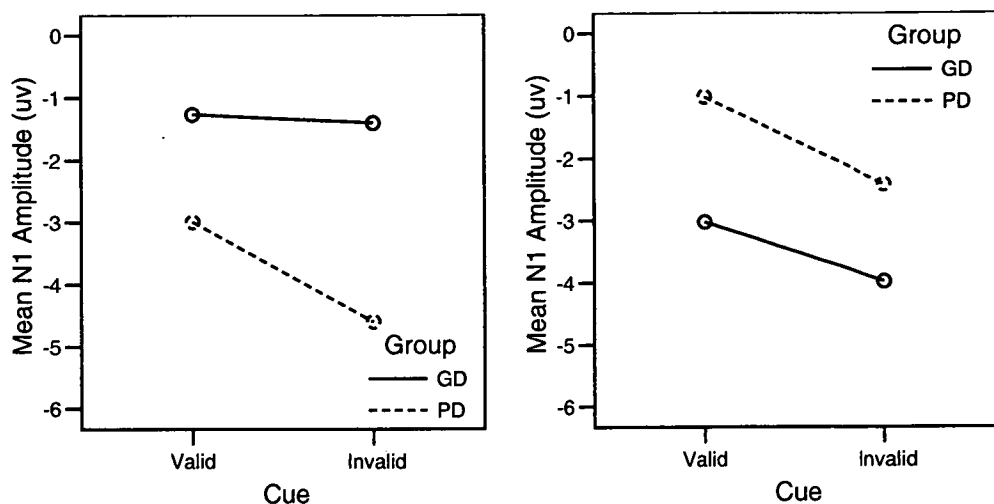


Figure 31. Mean N1 amplitude for R VF trials at ipsilateral (RH) parietal sites for the central task as a function of cue for female (left) and male (right) good decoders (GD) and poor decoders (PD).

N1 Latency

N1 latency was significantly shorter at parietal ($M=181.3$, $SEM=2.30$) in comparison to both occipital ($M=188.7$, $SEM=1.80$) and temporal ($M=187.6$, $SEM=1.93$) sites $F(1,35)=14.24$, $MSE=1436.1$, $p<.001$. N1 latency was significantly shorter at contralateral ($M=174.9$, $SEM=1.84$) in comparison to ipsilateral ($M=196.8$, $SEM=2.10$) sites, $F(1,35)=227.0$, $MSE=1894.4$, $p<.001$, for the 900ms ($M=184.4$, $SEM=1.91$) in comparison to the 400ms condition ($M=187.3$, $SEM=1.92$), $F(1,35)=6.50$, $MSE=1198.9$, $p=.015$, and for valid ($M=183.1$, $SEM=1.88$) in comparison to invalid trials ($M=188.6$, $SEM=1.99$), $F(1,35)=18.84$, $MSE=1476.9$, $p<.001$. The main effects of Task, VF, Group, and Sex were non-significant ($ps>.05$). There was a significant main effect of Hemisphere, $F(1,35)=18.68$, $MSE=1465.50$, $p<.001$, indicating shorter N1 latency in the LH ($M=183.1$, $SEM=1.70$) in comparison to the RH ($M=188.6$, $SEM=2.14$).

Figures 32-33 show a significant VF x Cue x Sex x Group interaction, $F(1,35)=10.93$, $MSE=346.9$, $p<.01$. The Cue x Sex x Group interaction was significant for LVF trials, $F(1,35)=6.16$, $MSE=45.73$, $p=.018$. For LVF trials, the overall effect of Cue was significant for females, $F(1,19)=14.40$, $MSE=48.59$, $p<.001$, and the Cue x Group interaction was significant for males, $F(1,16)=4.94$, $MSE=42.33$, $p<.05$, such the effect of Cue was significant for male good decoders, $F(1,16)=21.16$, $MSE=14.22$, $p<.01$, and not for male poor decoders ($ps>.05$). For RVF trials, the effect of Cue was significant for female good decoders, $F(1,9)=5.15$, $MSE=25.86$, $p<.05$, and poor decoders, $F(1,10)=10.99$, $MSE=36.10$, $p<.01$, but not for male good decoders or poor decoders ($ps>.05$). There were no significant between group differences ($ps>.05$).

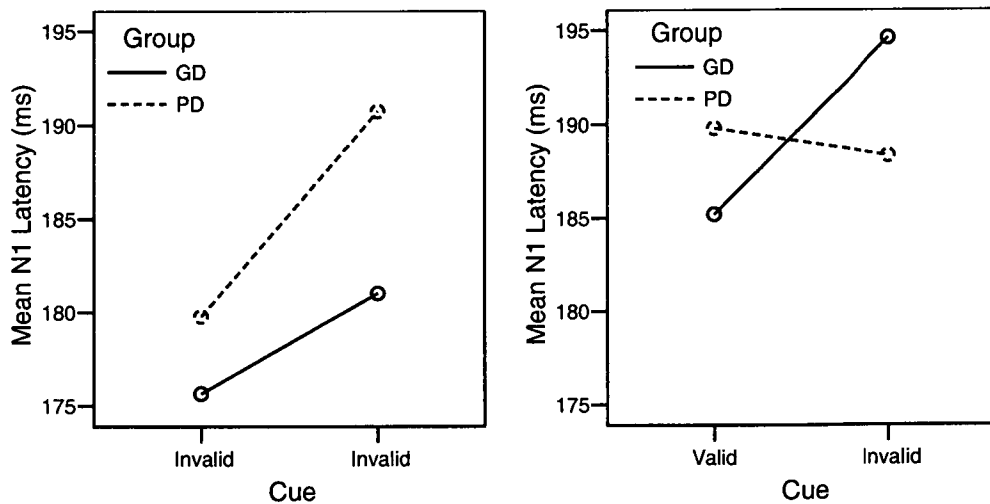


Figure 32. Mean N1 latency for LVF trials as a function of Cue for female (left) and male (right) good decoders (GD) and poor decoders (PD).

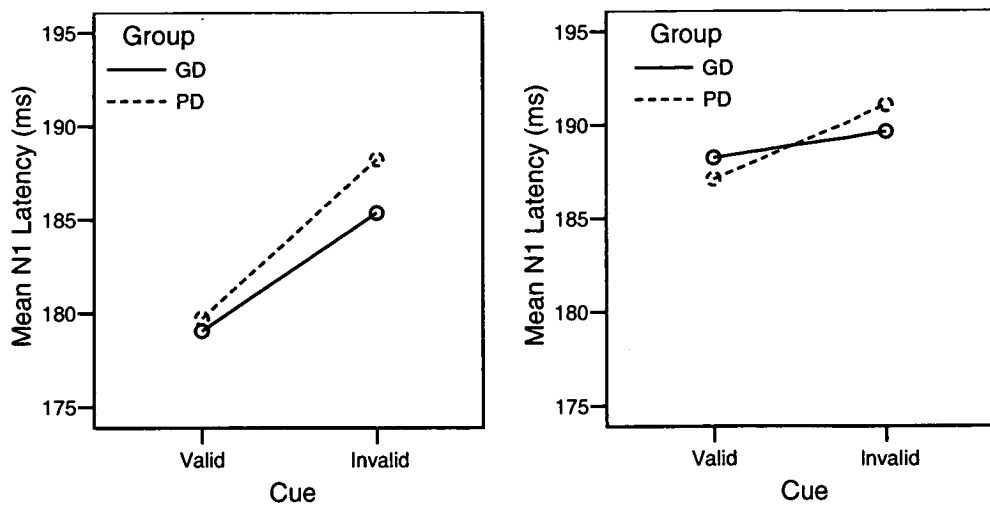


Figure 33. Mean N1 latency for RVF trials as a function of Cue for female (left) and male (right) good decoders (GD) and poor decoders (PD).

P1 Amplitude

P1 amplitude was significantly greater at parietal ($M=3.25$, $SEM=0.40$) in comparison to occipital ($M=2.50$, $SEM=0.28$) and temporal ($M=2.05$, $SEM=0.23$) sites respectively, $F(1,35)=12.76$, $MSE=45.6$, $p<.001$. P1 amplitude was significantly greater for the 900ms ($M=3.77$, $SEM=0.27$) in comparison to the 400ms ($M=1.44$, $SEM=0.35$) condition, $F(1,35)=68.0$, $MSE=74.3$, $p<.001$, at contralateral ($M=3.11$, $SEM=0.29$) in comparison to ipsilateral sites ($M=2.09$, $SEM=0.30$), $F(1,35)=25.81$, $MSE=37.8$, $p<.001$, and for the peripheral ($M=2.85$, $SEM=0.26$) in comparison to the central ($M=2.36$, $SEM=0.31$) task, $F(1,35)=12.12$, $MSE=18.5$, $p<.01$.

The main effect of Cue was significant, $F(1,35)=30.95$, $MSE=10.7$, $p<.001$, such that P1 amplitude was significantly greater for invalid ($M=2.90$, $SEM=0.31$) in comparison to valid trials ($M=2.30$, $SEM=0.26$). Figures 34-35 show a significant Task x Cue x Sex x Group interaction, $F(1,35)=8.54$, $MSE=9.36$, $p<.01$. For the central task, the overall effect of Cue was non-significant and did not interact with Sex or Group ($ps>.05$). However, for the peripheral task there was a significant Cue x Sex x Group interaction, $F(1,35)=8.54$, $MSE=9.36$, $p<.01$. The

overall effect of Cue was significant for females, $F(1,19)=34.00$, $MSE=12.16$, $p<.01$, and the Cue x Group interaction was significant for males, $F(1,16)=10.24$, $MSE=6.92$, $p<.01$, such that the effect of Cue was significant for male poor decoders, $F(1,8)=24.86$, $MSE=4.69$, $p<.01$, but not for male good decoders ($p>.05$). There were no significant effects of Group for males or females for either Task ($ps>.05$).

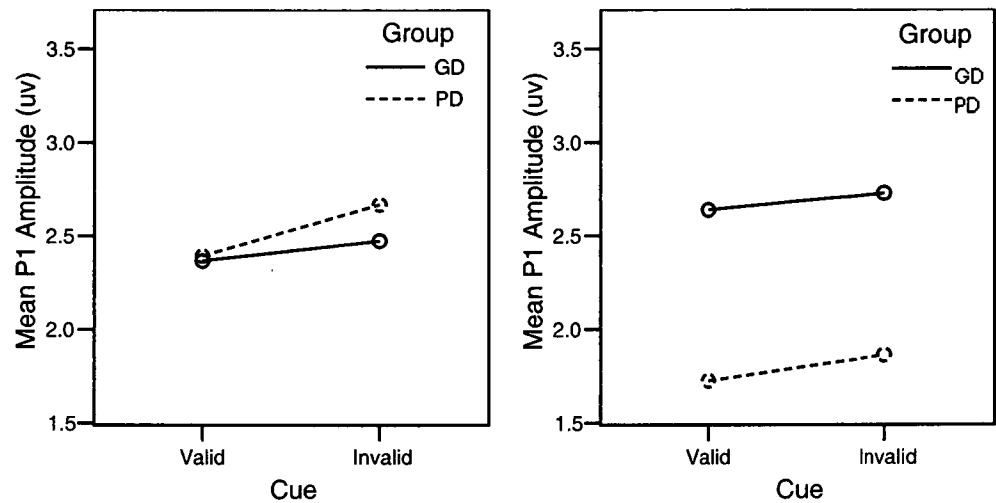


Figure 34. Mean P1 amplitude for the central task as a function of Cue for female (left) and male (right) good decoders (GD) and poor decoders (PD).

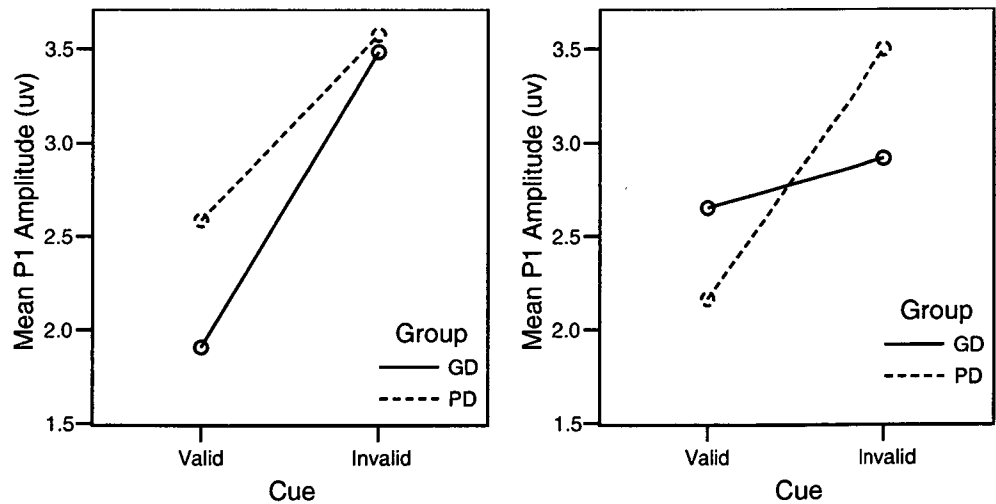


Figure 35. Mean P1 amplitude for the peripheral task as a function of Cue for female (left) and male (right) good decoders (GD) and poor decoders (PD).

For the peripheral task there was a significant Laterality x VF x Cue x Group interaction at parietal sites, $F(1,35)=8.86$, $MSE=0.716$, $p<.01$. At contralateral parietal sites there was a significant VF x Cue x Group interaction, $F(1,35)=6.14$, $MSE=1.04$, $p<.05$ (see Figure 36). Both groups showed a significant effect of Cue at ipsilateral sites. However, whereas good decoders showed a significant effect of Cue at contralateral sites for both LVF (RH) and RVF (LH) trials ($ps<.01$), poor decoders showed a significant effect of Cue for LVF (RH) ($p<.01$) but not RVF (LH) trials ($p>.05$), and there were no significant between group differences ($ps>.05$). However, it should be noted that among good decoders there was a significant Cue x Sex interaction for RVF trials at both contralateral, $F(1,17)=8.60$, $MSE=0.887$, $p<0.01$, and ipsilateral sites, $F(1,17)=5.62$, $MSE=1.34$, $p<0.05$, such that female good decoders showed a significant effect of Cue ($ps<.01$), and male good decoders did not ($ps>.05$). For poor decoders the effect of Cue was non-significant in the LH for RVF trials regardless of Sex ($ps>.05$).

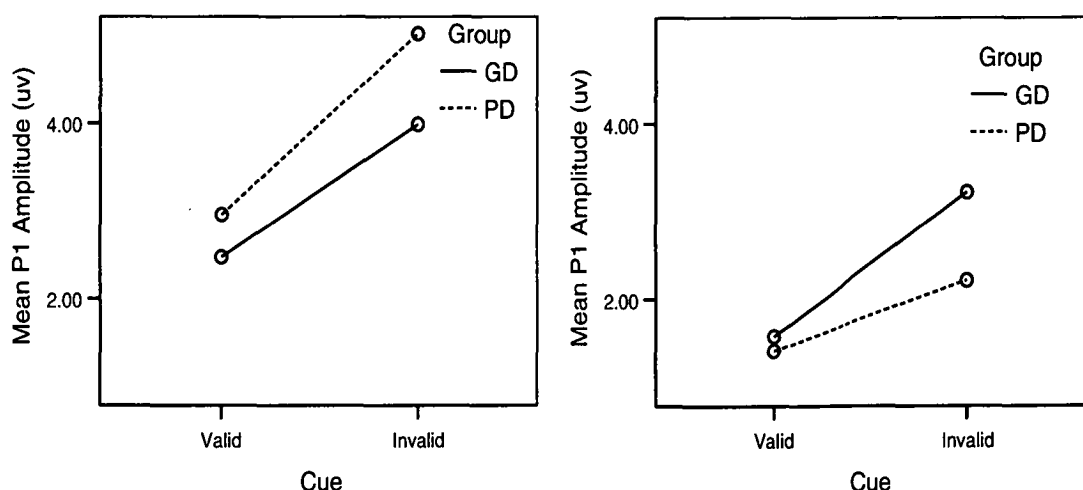


Figure 36. Mean P1 amplitude at contralateral parietal sites for good decoders (GD) and poor decoders (PD) as a function of cue validity for LVF (left) and RVF (right) trials for the peripheral task.

P1 Latency

There was a significant main effect of Sagittal site, $F(1,35)=5.48$, $MSE=348.6$, $p<.001$, indicating significantly greater P1 latency at parietal ($M=124.6$, $SEM=1.36$) and occipital ($M=123.6$, $SEM=1.11$) in comparison to temporal ($M=121.8$, $SEM=1.09$) sites. P1 latency was significantly shorter at contralateral ($M=113.0$, $SEM=1.20$) in comparison to ipsilateral ($M=133.7$, $SEM=1.22$) sites, $F(1,35)=348.6$, $MSE=1138.9$, $p<.001$, for the peripheral ($M=124.5$, $SEM=1.10$) in comparison to the central task ($M=122.5$, $SEM=1.18$) task, $F(1,35)=4.47$, $MSE=493.2$, $p<.05$, for the 400ms ($M=121.7$, $SEM=1.08$) in comparison to the 900ms ($M=124.9$, $SEM=1.20$) SOA condition, $F(1,35)=19.88$, $MSE=480.7$, $p<.001$. The main effect of Sex was significant, $F(1,35)=5.23$, $MSE=4339.2$, $p<.05$, indicating significantly shorter P1 latency for females ($M=120.8$, $SEM=1.5$) in comparison to males ($M=125.8$, $SEM=1.59$).

The main effect of Cue was significant, $F(1,35)=27.99$, $MSE=478.2$, $p<.001$, indicating significantly shorter P1 latency for valid ($M=121.4$, $SEM=1.1$) in comparison to invalid ($M=125.2$, $SEM=1.2$) trials. However, this was qualified by a significant Cue x Sex interaction, $F(1,35)=13.5$, $MSE=478.2$, $p<.01$, such that the overall effect of Cue was significant for females, $F(1,35)=45.94$, $MSE=452.9$, $p<.001$, but not for males ($p>.05$). Figures 37 and 38 shows a significant VF x Cue x Group interaction at parietal sites, $F(1,35)=15.2$, $MSE=153.8$, $p<.001$. For LVF trials, the overall effect of Cue was significant for both good decoders, $F(1,17)=14.36$, $MSE=37.63$, $p<.01$, and poor decoders $F(1,18)=8.73$, $MSE=20.23$, $p<.01$. However, for RVF trials there was a significant Cue x Group interaction, $F(1,17)=8.99$, $MSE=31.58$, $p<.01$, such that the effect of Cue was significant for poor decoders, $F(1,18)=22.21$, $MSE=46.02$, $p<.001$, and only approached significance for good decoders ($p=.073$). This effect was largely due to a significant Cue x Sex interaction for good decoders, $F(1,17)=18.72$, $MSE=16.28$, $p<.001$, such that the effect of Cue was significant for female good decoders ($p<.001$) and not for male good decoders ($p>.05$). Further, for RVF trials, there was a significant Cue x Group interaction for males, $F(1,16)=8.51$, $MSE=37.66$, $p<.01$, such that the effect of Cue approached significance for

male poor decoders ($p=.037, p>.05$, Bonferroni corrected) and was non-significant for male good decoders ($p>.05$). Male good decoders tended to show shorter P1 latency in comparison to male good decoders for RVF invalid trials, $F(1,16)=3.17, MSE=153.6, p=.094$. There were no other between group differences ($ps>.05$).

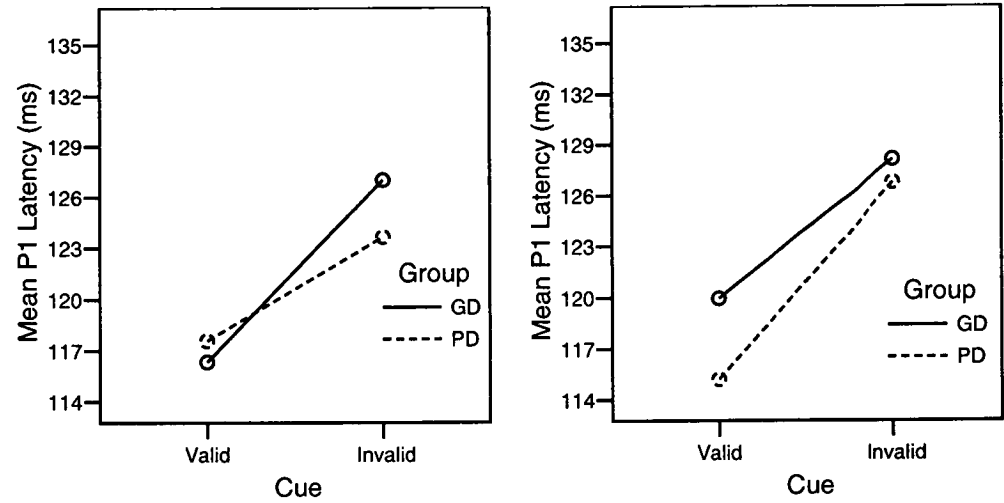


Figure 37. Mean P1 latency at parietal sites for female good decoders (GD) and poor decoders (PD) as a function of Cue for LVF (left) and RVF (right) trials.

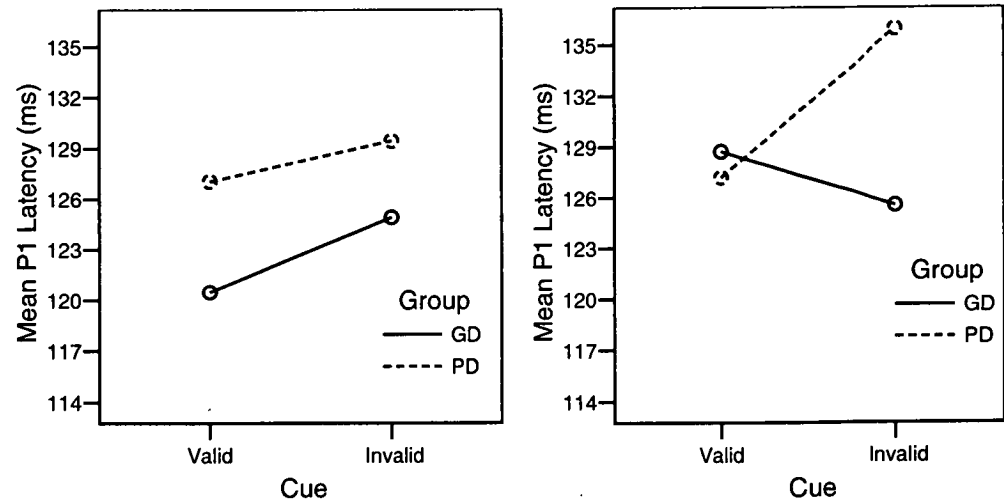


Figure 38. Mean P1 latency at parietal sites for male good decoders (GD) and poor decoders (PD) as a function of Cue for LVF (left) and RVF (right) trials.

Discussion

Adult poor phonological decoders tended to show longer RT overall in comparison to good phonological decoders. This is consistent with previous research showing that dyslexic children are slower than normally reading controls when responding to targets preceded by both central and peripheral cues (Facoetti et al., 2003b; Facoetti et al., 2003c; Facoetti et al., 2000b; Jonkman et al., 1992). As predicted, RT was significantly shorter for valid in comparison to invalid trials. Although both good and poor decoders showed a significant effect of cue validity, poor decoders tended to show longer RT for valid trials in which cues correctly predicted the location of subsequent targets. This indicates that poor decoders showed less benefit of early spatial selection. Females showed a greater effect of cue validity on RT in comparison to males, however, this was largely due to the lack of a spatial cueing effect for male poor decoders. Further, the RT benefit analysis (neutral minus valid) performed on the data showed that good decoders showed greater benefits in comparison to poor decoders, and this effect was greatest for males.

These findings are consistent with previous findings indicating that dyslexics (Brannan & Williams, 1987) and poor readers (Kinsey et al., 2004) benefit less from valid spatial cues. This group difference in the benefits of spatial attention was also greater for the central task relative to the peripheral task. Examination of VF differences revealed that the reduction in RT benefit of valid cues for poor decoders was greater in the LVF. This effect was also found to be greater for males, with male good decoders but not poor decoders showing an overall effect of cue for LVF trials. The analysis of RT costs (neutral minus invalid trials) also revealed that poor decoders showed greater costs for the central relative to the peripheral task when stimuli were presented in the LVF. A disruption of spatial cuing in the LVF is consistent with previous research reporting LVF inattention or mini-neglect in dyslexic populations and may be related to functioning of RH posterior parietal areas (Eden et al., 2003; Facoetti & Turatto, 2000; Hari et al., 2001). Unilateral spatial neglect as a consequence of RH parietal damage can result in

reduced ability to report the left side of pages, lines, or words during reading. Further, patients with right posterior lesions are able to identify words correctly but fail to report letters on the contralesional (left) side of nonwords when attentional demands are greatest (Brunn & Farah, 1991; Sieroff et al., 1988).

For RVF trials, poor decoders showed an overall increase in RT relative to good decoders. There was a significant RVF advantage for the peripheral cueing task; however, this was largely due to good decoders who tended to show shorter RT for RVF trials in comparison to poor decoders. Whereas few previous studies have reported a RVF advantage for peripheral cueing tasks, the letter discrimination task used in the present study may have increased the involvement of LH language areas, producing a contralateral RVF advantage. Previous research findings indicate that dyslexics often fail to show the same right visual field advantage as controls (Bloch & Zaidel, 1996; Kershner, 1977; Marcel et al., 1974; Pirozzolo & Rayner, 1979). Further, Brannan and Williams (1987) found that good readers and adults but not poor readers showed a significant RVF accuracy advantage in a go/nogo peripheral cueing experiment requiring the detection of letters.

Overall accuracy was unaffected by spatial cue manipulations and there were no overall accuracy differences between good and poor phonological decoders. There was however some evidence for a speed/accuracy trade-off among males for the central task. Male good decoders were less accurate in comparison to poor decoders for the central task overall but showed greater benefits of cueing manipulations on RT for this task. However, mean accuracy was above 80% for both groups. Male good decoders also showed a reduction in accuracy for RVF invalid trials relative to male poor decoders but there was no corresponding trade-off in RT.

The effect of spatial cueing on N1 amplitude was significant at contralateral sites for both the central and peripheral tasks. For the central task, females and male good decoders showed N1 modulation for LVF (RH) trials and male poor decoders did not. This finding is consistent with the RT data and indicates that the reduction in spatial cueing in the LVF among

male poor decoders was coupled with a lack of attentional modulation of the N1 component in RH parietal areas. The reduction in N1 amplitude among male poor decoders is also consistent with the findings of Experiments 2 and 3 in the current series of experiments. Females showed a greater cueing effect for RVF trials in comparison to males at LH parietal sites. For the peripheral task, the effect of cue was significant for females but not males for LVF (RH) trials, and for RVF (LH) trials, the effect of cue was greater for female good decoders in comparison to female poor decoders, and a similar trend was observed for males.

Good decoders showed greater N1 amplitude in the ipsilateral LH for LVF trials in comparison to poor decoders at both temporal and parietal sites. At temporal sites, this difference was largely due to an increase in N1 amplitude for valid trials for female good decoders and an increase in N1 amplitude on invalid trials for male good decoders. At ipsilateral parietal sites there was an overall reversal of the spatial cueing effect on N1 amplitude for the peripheral task, such that N1 amplitude was significantly greater for invalid in comparison to valid trials. However, the effect of cue was largely due to a significant effect for male good decoders for LVF (LH) trials. Some previous research has also found evidence for an ipsilateral invalid negativity, which may be related to the mechanism of disengaging attention which originates in temporo-parietal areas (Hopfinger & Mangun, 2001). For example, patients with lesions to the posterior temporal gyrus (temporal-parietal junction) are slower to respond to invalid targets that appear contralateral to the lesion (Hopfinger & Mangun, 2001). The present findings indicate that whereas male good decoders showed less facilitation for valid trials as indexed by N1 amplitude modulation for the peripheral task, they showed greater evidence for the disengagement of attention on invalid trials, particularly when attention was previously engaged in the RVF.

P1 amplitude was maximal at contralateral parieto-occipital sites, and was modulated by attention for the peripheral task such that amplitude was significantly greater for invalid in comparison to valid trials. This is consistent with previous research showing a reversal of the P1

attention effect in peripheral cueing paradigms, and particularly those that have shown a P1 reduction effect in the absence of behavioural inhibition of return during two-choice discrimination tasks (Eimer, 1994; Hopfinger & Mangun, 1998). For the peripheral task, both groups showed a significant effect of Cue at ipsilateral parietal sites. However, whereas good decoders showed an effect of Cue at contralateral sites for both LVF (RH) and RVF (LH) trials, poor decoders showed an effect of Cue for LVF (RH) but not RVF (LH) trials. However, it is likely that the effect of peripheral cues on P1 amplitude among good decoders was largely attributable to the responses of female good decoders, as the overall P1 cueing effect was not significant for male good decoders. Behavioural inhibition of return may be absent in the presence of P1 reduction suggesting that inhibitory (P1) and excitatory (N1) effects compete and have a differential influence over time (Mangun, 1995). Thus the present findings indicate that male good decoders experienced less perceptual inhibition during the performance of the peripheral cueing task overall and that poor decoders as a group showed less P1 modulation in the LH for RVF trials. It is possible that these findings contribute to the lack of a RVF RT advantage seen among poor decoders.

Both P1 and N1 latency were modulated by attention in the present study such that latency was shorter for valid than invalid trials. Although not a common finding, some previous studies have also observed attentional modulation of P1 and N1 latency (Anllo-Vento & Hillyard, 1995; Wascher & Tipper, 2004). In the present study, N1 latency tended to be shorter for females in comparison to males overall and for LVF trials the effect of Cue was significant for females and male good decoders, but not for male poor decoders. This is consistent with the RT data indicating fewer benefits of valid spatial cues for male poor decoders relative to the other groups, particularly for LVF trials. The modulation of P1 latency by attention in the present study was found to be significant for females and not for males. However, this interacted with group and VF at parietal sites such that male good decoders tended to show a reduction in P1 latency for RVF invalid trials relative to male poor decoders.

Although previous research has shown that the behavioural cueing effects of dyslexic children are greatest for peripheral cueing tasks with short cue-to-target SOAs, suggesting an automatic orienting deficit (Brannan & Williams, 1987; Facoetti et al., 2000b; Heiervang & Hugdahl, 2003), the present study has demonstrated differences between adult good and poor phonological decoders on both central and peripheral cueing tasks with a relatively long cue-to-target SOA. Further, whereas Facoetti et al. (2001) found that dyslexic children showed a greater effect of cue in the LVF than the RVF, the reverse pattern was found in the present study. The present study differs from many previous studies as a two-choice letter discrimination task was used rather than simple target detection task. The voluntary allocation of attention during discrimination tasks and particularly those that employ letter stimuli may be more similar to the process of reading, whereas the voluntary allocation of attention during target detection tasks is likely to involve different interactions between the posterior and anterior attention systems. It is also possible that compensation strategies developed by the adult poor decoders in the present study contributed to discrepancies with previous research.

The present study was not designed to investigate sex differences and may lack the statistical power to reveal reliable differences, as indicated by the large number of trends in the data. Although these preliminary findings should be interpreted with caution and require further investigation, females tended to show greater effects of spatial attention in comparison to males. For example, for the central task females but not males showed attentional modulation in the contralateral LH for RVF trials and for the peripheral task females showed a greater effect of cue at contralateral sites relative to males. Previous research has also shown that females show greater cueing effects in comparison to males in a letter discrimination task in which targets were preceded by uninformative central cues, while no sex differences were observed for an exogenous orienting task (Bayliss, di Pellegrino, & Tipper, 2005). Previous research also indicates that dyslexic females show a larger orienting effect relative to dyslexic males (Bednarek et al., 2004).

Most of the previous studies reporting behavioural differences between dyslexic and control children on covert orienting tasks have included only male participants or have included a greater proportion of males in comparison to females (Facoetti et al., 2003b; Facoetti et al., 2003c; Facoetti et al., 2000b; Facoetti et al., 2001). Thus, it is possible that the deficits in spatial attention observed among dyslexic populations are largely due to the performance of male dyslexics. In the present study, male poor decoders showed less behavioural benefits of attention, coupled with less attentional modulation of the N1 component in the RH and less attentional modulation of N1 latency. These findings are broadly consistent with the RH parietal deficit hypothesis of dyslexia.

Whereas male poor decoders showed less attentional modulation in the RH, the present findings indicate that female poor decoders differed most from female good decoders in terms of overall lateralisation of ERP components. For example, female good decoders showed a greater effect of cue at LH parietal sites for RVF trials relative to female poor decoders and at ipsilateral LH temporal sites for LVF trials, suggesting greater attentional modulation in the LH relative to female good decoders. Instead female poor decoders showed greater N1 amplitude in the ipsilateral RH for RVF trials suggestive of compensatory processing.

It should also be noted that the male good decoders in the present study differed from the other groups on a number of behavioural and electrophysiological indices. For example, male good decoders were less accurate for the central task overall and for RVF invalid trials relative to male poor decoders. Male good decoders also showed greater N1 amplitude modulation (invalid greater than valid trials) in the ipsilateral LH for LVF trials relative to other groups, less P1 amplitude modulation (invalid greater than valid trials) for the peripheral task (particularly for RVF trials), and a reduction in P1 latency for RVF invalid trials. These effects were typically unexpected and further research is required to assess whether male and female good decoders differ consistently on these measures or whether other factors such as motivation or strategy use can account for these sex differences among the good readers.

Based on the spatial orienting deficits observed among dyslexic populations it has been argued that both the facilitation of processing at attended areas and reduction of interactions from surrounding visual stimuli is important for the letter parsing and segmentation necessary during the phonological decoding of words (Facoetti et al., 2005). Consistent with this hypothesis, the present study has shown that adults who are poor phonological decoders also show differences in spatial attention relative to good decoders. Specifically poor decoders showed less RT benefit of valid spatial cues relative to good decoders, particularly for LVF trials. This effect was greatest for male poor decoders, who also showed a lack of N1 modulation in the RH for LVF trials and an overall lack of attentional modulation of N1 latency. Female good decoders showed greater N1 modulation in the ipsilateral LH for LVF trials. In contrast, female poor decoders showed greater N1 amplitude in the ipsilateral RH for RVF trials indicative of compensatory processing. Although further research is required to assess the reliability of these sex differences, preliminary evidence suggests that spatial attention deficits among poor readers may be greater for males in comparison to females. The aim of following experiment was to further investigate covert orienting differences between good and poor decoders during the performance of cued orthographic and phonological decision tasks.

CHAPTER 10 - EXPERIMENT 5: PHONOLOGICAL DECODING ABILITY AND
ELECTROPHYSIOLOGICAL INDICES OF SPATIAL ATTENTION DURING CUED
PHONOLOGICAL AND ORTHOGRAPHIC DECISION TASKS.

Lexical decision tasks require participants to make decisions about a pair of bilaterally presented words or single words presented unilaterally to either visual field. In tasks in which participants decide whether a letter string is a real word or a nonword, lexicality effects are observed such that shorter RT and greater accuracy are observed for real words relative to nonwords (McCann et al., 1992). The RVF advantage in accuracy and/or decision time observed during the performance of lateralised lexical decision tasks (Nicholls & Wood, 1998; Ortells, Tudela, Noguera, & Abad, 1998) has been attributed to the functional specialisation of the contralateral LH for processing language (Kimura, 1961). According to the “callosal relay” model, the RVF advantage reflects slowing down or degradation of information processing due to callosal relay from the RH to the LH. In contrast, the “direct access” model suggests that the RH is less effective at processing linguistic information and may use a different strategy than the LH (Zaidel, 1983). According to other attentional theories the presentation of verbal stimuli activates the LH resulting in a rightward attentional bias (Kinsbourne, 1970), or the LH requires fewer attentional resources in order to process linguistic stimuli relative to the RH (Mondor & Bryden, 1992). It has also been suggested that the LH uses a parallel strategy whereas the RH uses a serial strategy, suggesting specialisation for whole word recognition and sequential sublexical processing respectively (Chiarello, 1988; Lindell & Nicholls, 2003).

The relationship between spatial attention and word recognition can be investigated by examining the effect of spatial cuing procedures on lexical decisions. Some studies have found that spatial cues do not influence lexical decision performance (Hardyck, Chiarello, Dronkers, & Simpson, 1985). These findings are consistent with late selection accounts which posit that orthographic, phonological, and semantic codes are activated relatively automatically regardless

of spatial attention (LaBerge & Samuels, 1974). However, using traditional covert orienting paradigms in which cues predict whether words appear in the LVF or RVF consistent spatial attention effects have been found that do not interact with word familiarity (McCann et al., 1992; Ortells et al., 1998). This finding supports early selection accounts of word recognition and suggests that spatial attention is allocated to letter strings prior to identification (Treisman, 1988; Treisman & Gelade, 1980). Whereas the tasks used in these studies required nonword/real word lexical decisions and a motor response, other researchers have investigated the effects of spatial attention on word naming latencies. The results of these studies are typically consistent with familiarity sensitive models of attention in reading (LaBerge & Brown, 1989; Mozer & Behrmann, 1990). For example, a brief visual cue near the beginning or end of letter strings affects naming accuracy of centrally presented unpronounceable nonwords to a greater extent than pseudowords and real words respectively (Auclair & Sieroff, 2002; Sieroff & Posner, 1988). According to the attentional redistribution hypothesis, the lexical status of a letter string influences the distribution of spatial attention during and/or prior to word recognition (Auclair & Sieroff, 2002). In accordance with the dual route conceptualisation of word recognition, it has also been argued that reading via the nonlexical route requires the left to right allocation of spatial attention, whereas reading via the lexical route is a parallel process (Cestnick & Coltheart, 1999).

Ortells et al. (1998) investigated the influence of spatial attention on word/nonword lexical decisions by manipulating SOA and word frequency under central and peripheral cueing conditions in the presence or absence of distractor stimuli. All attentional effects were significant and did not interact with VF or word familiarity providing further evidence that attentional orienting facilitates word recognition. McCann et al. (1992) investigated the effects of peripheral spatial cues on lexical decisions to high and low frequency words and pronounceable nonwords presented above or below fixation. There was an overall effect of spatial cue that did not interact with either word frequency or word type, suggesting an additive effect on processing occurring

prior to lexical access. McCann et al. (1992) proposed that additive effects of spatial attention on word recognition performance can be explained by Type-1 early selection in which attention is allocated to the cued location and must be repositioned on invalid trials prior to lexical access (e.g., Posner, Snyder, & Davidson, 1980a) or Type-2 accounts in which the efficiency of processing (e.g., feature extraction) is reduced when attention is cued to other locations (Hawkins, Shafto, & Richardson, 1988, cited in McCann et al., 1992). To investigate further whether word recognition and spatial attention operate sequentially and independently, Stolz and McCann (2000) combined an exogenous cueing procedure with a semantic priming paradigm. Spatial cueing was found to have a smaller effect for related relative to unrelated prime-target pairs, and semantic priming effects were greater for invalid relative to valid trials. These over-additive effects disappeared when cue validity was reduced from 80% to 50%. Together these findings suggest an overlap in the processes involved in both spatial orienting and word recognition. It was suggested that spatial attention affects the uptake of information in the orthographic input lexicon, and may also determine whether semantic information feeds down to the orthographic level (Stolz & McCann, 2000).

There is some evidence for an interaction between spatial cueing, visual field, and word familiarity that is consistent with hemispheric differences in lexical processing. For example, findings of a RVF advantage for words but not for nonwords (Hardyck et al., 1985; Ortells et al., 1998) are consistent with the proposal that the LH and RH use different strategies that are specialised for parallel lexical and serial sublexical processing respectively (Chiarello, 1988). Spatial cueing has also been shown to have a greater effect on both real word and nonword naming latencies when words are presented in the LVF rather than the RVF (Gatheron & Sieroff, 1999; Lindell & Nicholls, 2003; Nicholls & Wood, 1998). Gatheron and Sieroff (1999) found a significant effect of peripheral spatial cues on naming latencies of both words and nonwords in the LVF but not RVF. Nicholls and Wood (1998) found that peripheral cues affected accuracy and naming latency of high and low frequency words presented in the LVF but not the RVF.

Consistent with the attentional advantage model (Mondor & Bryden, 1992), the RVF advantage was effectively reduced and accentuated when attention was directed or misdirected respectively. Similarly, cues at either the beginning or the end of words affect naming latency of words in the LVF but not RVF (Lindell & Nicholls, 2003). In accordance with Mondor and Bryden (1992), it was argued that the LH distributes attention over the entire word facilitating parallel lexical processing, whereas the sequential word recognition strategy of the RH is more attentionally demanding and affected by spatial attention (Lindell & Nicholls, 2003). It has recently been suggested that word recognition by the LH involves parallel bottom-up activation of lexical representations, whereas word recognition by the RH involves the transformation of letters into abstract letter identities followed by grapheme-phoneme conversion in the LH and top-down support from the word to the letter level and therefore an extra visuo-orthographic stage prior to lexical access (Ellis, 2004).

The present study employs lexical decision tasks that bias processing towards either whole word recognition, and therefore the parallel allocation of attention to letter strings (orthographic decision task), or recognition based on the phonological representation of words and therefore the serial allocation of attention to letters within letter strings (phonological decision task). Phonological decision tasks require participants to decide which of two nonwords is a homophone of a real word (e.g., *kake*, *dake*) (Olson et al., 1985; Stanovich & Siegel, 1994). This task requires the generation of sound codes for nonwords that do not have existing lexical representations and therefore requires phonological recoding. In contrast, orthographic decision tasks require participants to decide which of two homophones is a real word (e.g., *rume*, *room*) (Olson et al., 1985; Stanovich & Siegel, 1994). In this case, the words have the same phonological code and decisions are based on orthographic information and access to existing lexical representations (Olson et al., 1985). Decision times are typically longer for phonological relative to orthographic decision tasks, suggesting greater demands on resources and/or slower speed of processing, which may be related to the sequential nature of phonological processing

(Breznitz, 2003). Consistent with the phonological basis of dyslexia, dyslexic children typically show greater impairment on phonological relative to orthographic decision tasks when compared to controls (Olson et al., 1985; Stanovich & Siegel, 1994). Further, it has also been found that whereas dyslexic children showed lower performance for both real words and pseudowords on a lexical decision task, adult dyslexics were only impaired relative to controls for pseudoword or phonological processing (Breznitz, 2003; Breznitz & Misra, 2003). No previous study has investigated the relationship between reading ability and the effects of spatial attention on lexical decisions that are biased towards either phonological or orthographic processing.

The present study employed a modified version of the phonological and orthographic lexical decision tasks outlined above in which words were presented unilaterally to either the LVF or RVF and were preceded by valid or invalid central spatial cues. Poor phonological decoders are expected to be slower and less accurate in comparison to good phonological decoders. Due to the selection of participants based on phonological decoding ability differences are expected to be greatest for the phonological relative to the orthographic task (Breznitz, 2003; Breznitz & Misra, 2003; Olson et al., 1985; Stanovich & Siegel, 1994). A RT advantage is expected for valid in comparison to invalid spatial cues. If spatial attention has its effect prior to lexical access, no interaction is expected between the effect of spatial attention and task (Ortells et al., 1998), however, if spatial attention interacts with word familiarity, the effect of spatial attention is expected to be greater for the phonological relative to the orthographic task. Several previous studies have shown that dyslexics differ from normal readers in the covert orienting of spatial attention (Brannan & Williams, 1987; Facoetti et al., 2001), and findings from the present series of experiments suggest that adult poor phonological decoders differ from good phonological decoders on these tasks. Considering that phonological decoding requires the sequential allocation of attention to letters, group differences in the effects of spatial attention are expected to be greatest for the phonological task. Further, some studies have found evidence for LVF mini-neglect in dyslexia suggestive of a RH parietal deficit (Eden et al., 2003; Facoetti

& Turatto, 2000; Hari et al., 2001). The findings of Experiment 4 in the present series of experiments indicate that poor phonological decoders (particularly males) show a similar deficit. Thus, in the present study, poor decoders are expected to show disrupted spatial attention for LVF trials, particularly for the phonological task which requires the sequential allocation of attention.

According to Vidyasagar (1999, 2001, 2004), dyslexia stems from a deficient spotlighting mechanism that originates in the PPC and affects subsequent processing in the ventral visual stream. A large body of electrophysiological research has shown that selective visual attention to spatial locations modulates the early visual components of the ERP waveform (see Mangun, 1995). However, few electrophysiological studies have directly investigated the link between spatial attention and reading disability. Attentional manipulations have been shown to modulate the early visual components of the ERP waveform. Thus a further aim of the present study is to investigate the time course and functional properties of spatial attention during orthographic and phonological decision tasks by comparing ERP waveforms elicited by words preceded by valid or invalid spatial cues. Attention to specific spatial locations results in increased P1 and N1 amplitude to stimuli in that region, particularly at posterior contralateral sites (for reviews see Eimer, 1998; Mangun, 1995). It has been suggested that spatial selection acts to decrease signal to noise ratio of inputs in the visual field, facilitating processing of attended locations at higher stages of perceptual processing (Mangun, 1995). Differences in the modulation of the P1 and N1 components as a function of task demands have been discussed in detail in Chapter 9 of the present thesis.

Breznitz (2003) found no differences in N1 amplitude or latency between male adult dyslexics and controls for orthographic and phonological decision tasks, but dyslexics showed greater N1 amplitude at midline central sites (Cz) for homophone pairs in an orthographic-phonological choice task in which participants decided whether homophone or homograph pairs sounded alike or looked alike. Wimmer et al. (2002) found a reduction in N1 amplitude at RH

sites for words but not pseudowords during a word naming task. However, no previous study has investigated the modulation of the P1 and N1 components by attentional manipulations during lexical decisions. If the early P1 and N1 components are modulated by attention in the present study, greater amplitude and shorter latencies are expected for valid relative to invalid trials. A reduction in the attentional modulation of these components among poor phonological decoders would indicate differences in early spatial selective attention. Further, if differences are greatest in RH parietal areas this would be consistent with the RH deficit hypothesis of dyslexia.

Method

Participants

This study was approved the University of Tasmania Human Research Ethics Committee. All participants gave written informed consent prior to participation in the study. Exclusion criteria included a history of drug, alcohol, or tobacco abuse, psychiatric or neurological disorder, head trauma, seizure, and those currently receiving medication. Twenty six first year psychology students at the University of Tasmania participated in this experiment as part of their course requirement. Good ($n=13$, 8 female, 5 male) and poor ($n=13$, 9 female, 4 male) phonological decoders were selected from a larger sample on the basis of Martin and Pratt Nonword Reading Test scores (Martin & Pratt, 2001). . Two participants in each group were left-handed, and the remaining participants were right handed as measured by the Edinburgh handedness inventory (Oldfield, 1971). All participants had normal or corrected to normal vision. The scores of good phonological decoders ranged from 49-54 (out of a possible score of 54) and the scores of poor phonological decoders ranged from 19-41. Norms for the Nonword reading test are available for samples up to 17 years of age (Martin & Pratt, 2001). The mean score of good decoder group was in the 81st percentile (> 17 years reading age equivalent) and the mean score of poor decoders was in the 18% percentile (10-11 years reading age equivalent) of this norming group.

Several reading and neuropsychological measures were administered in a screening session of approximately one hour on a day prior to the experimental session. Ravens Advanced Progressive Matrices (APM) was administered as a measure of non-verbal general intelligence (Raven et al., 1994). Other reading measures included the Word Identification and Comprehension subtests from the Woodcock-Johnson (WJ) Reading Mastery Tests (Woodcock, 1987), The National Adult Reading Test (NART: Nelson & Willison, 1991), an irregular word reading test (see Appendix A), and Reading Accuracy and Reading Rate measures from the Neale Analysis of Reading Ability. Other measures included the Digit Span, Vocabulary, Symbol Coding and Symbol Copy sub-tests from the Weschler Adult Intelligence Scale (WAIS-III: Wechsler, 1997).

Table 6 shows mean age, APM raw score, and scores on reading measures for each group. Good readers were significantly older than poor readers, but ages ranged from 18 to 24 years and there was no significant group difference in nonverbal intelligence as measured by APM raw scores. Poor readers had significantly lower scores on measures of nonword reading, word identification and irregular word reading (irregular word reading and NART scores). Due to the selection of groups based on nonword reading ability, there was a ceiling effect for good readers on this measure. The scores of poor readers were more variable with larger standard deviations in comparison to good readers for measures of nonword reading, word identification, irregular word reading, vocabulary, and symbol coding. Poor readers showed significantly lower scores in comparison to good readers on digit span forwards but not backwards, indicating a difference in verbal short term memory but not necessarily attention and concentration. There was a trend for good decoders to have higher scores on the symbol search subtest than poor decoders. This is consistent with literature showing concurrent linguistic and short term memory deficits in dyslexic populations (e.g., Jorm, 1983). Poor readers also showed poorer comprehension in comparison to good readers as measured by the passage completion subtest of

the Woodcock Reading Mastery tests and lower accuracy and reading rate as measured by the Neale analysis of reading ability.

Table 6

Mean age and raw scores on reading and cognitive measures for good and poor phonological decoders in Experiment 5.

	Good Decoders		Poor Decoders	Sig.	
	(n=13)		(n=13)		
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	
Age	21.9	3.78	19.2	1.49	*
APM /36	24.0	5.26	21.1	3.35	<i>ns</i>
MP Nonword reading /54	50.6	1.61	34.3	6.66	***
WJ Word Identification /106	99.2	1.83	86.9	5.11	***
Irregular word reading /87	81.9	2.94	60.0	7.78	***
NART [#] /50	35.5	2.90	18.00	4.56	***
DS Forwards [#] /16	11.8	1.92	9.00	1.68	**
DS Backwards [#] /14	8.08	1.71	7.00	2.08	<i>ns</i>
WJ Comprehension /68	61.5	2.5	56.4	4.7	**
WAIS Vocabulary [†] /66	56.5	5.2	43.2	8.0	***
WAIS Symbol coding [†] /60	82.2	8.2	74.5	8.8	*
WAIS Symbol copy [†] /133	125.4	9.8	114.5	19.1	<i>p</i> =.078
Neale Reading Accuracy [†] (%)	98.0	2.35	87.2	5.48	***
Neale Reading Rate [†] (words/min)	147.8	17.8	110.3	11.49	***

Note: * $p < .05$, ** $p < .01$, *** $p < .001$, [#] Missing data was substituted with the mean for the group for one poor decoder, [†] Missing data was substituted with the mean for the group for one poor decoder and three good decoders.

Stimuli and Apparatus

Stimuli were presented on an IBM computer and tasks were programmed using the NeuroScan STIM program. The phonological and orthographic decision tasks were modified versions of the phonological and orthographic coding tasks used by Olson et al. (1985). However, the task was modified such that single rather than pairs of words required a decision. Two word lists of 120 four or five letter words were constructed (see Appendix B) for the orthographic decision task (real words/pseudohomophones) and the phonological decision task (pseudohomophones/nonwords). Words lists included the words used by Olson et al. (1985) with additional real words chosen from the Kucera-Francis noun database (Kucera & Francis, 1970) and pseudohomophones and nonwords from the ARC Nonword database (Rastle, Harrington, & Coltheart, 2002).

For the orthographic decision task (ODT), the frequency of the real words ranged from 1 to 275 with an average frequency of 44.5, and each real word was matched with a pseudohomophone with the same phonological representation. For the phonological decision task (PDT), non words were matched by changing either the onset or rime of each word in a list of pseudohomophones to create a new pronounceable nonword with no existing phonological representation in the English language. All pseudohomophones and nonwords were chosen from the ARC Nonword database and consisted of orthographically existing onsets and bodies and legal bigrams and monomorphemic syllables only.

Each trial began with a 200ms presentation of a central arrow cue pointing to either the left or right visual field. Word stimuli were presented for 1500ms in the left or right visual hemifield with an SOA of 500ms after the offset of the arrow. The inter-trial interval was 1000ms. The cue stimulus subtended 1 x 1 degrees of visual angle at a viewing distance of 70cm. On 75% of trials, cues were directionally informative and the target was presented to the visual field indicated by the cue. On 25% of trials the target was presented contralateral to the cued location (invalid). Each task consisted of 240 trials (192 valid, 48 invalid). Word stimuli

were horizontal lowercase letter strings presented white on a black background. Each letter subtended 1 x 1 degrees of visual angle at a viewing distance of 70cm. The inner edge of each word stimulus was presented 6 degrees to the right or left of fixation. Within each task word types were presented randomly.

Electrophysiological Recording

EEG activity was recorded with a NeuroScan system, consisting of a 32-channel Synamps, SCAN 4.1 software, and Quik-cap with Ag/AgCl electrodes interfaced with a NeuroScan STIM 3.1 computer. EEG was recorded from 32 sites, according to the conventional 10-20 system, and all electrodes were referenced to the mastoids. Horizontal electro-oculographic (EOG) activity was recorded bipolarly from electrodes at the outer canthi of both eyes, and vertical EOG was recorded from electrodes above and below the left eye. Electrode impedance was kept below 5 k Ω . EEG activity was amplified with a bandpass of 0.15-100 Hz, and sampled continuously at a rate of 1000 Hz. EEG data were merged with behavioural files and ocular artefact reduction was conducted by regression and artifact averaging (Semlitsch, Anderer, Schuster, and Presslich, 1986). Data files were epoched offline for a 1000ms epoch commencing 100ms before stimulus onset and were baseline corrected. High and low voltage cut-offs for artefact rejection were set at 100 μ V and -100 μ V respectively. Correct responses for each word-type were averaged and then band-pass filtered (0.5-30 Hz). Posterior P1 and N1 components were determined from grand averaged means as the maximum voltage within the following time frames after word onset: (P1: 80-150ms; N1: 150-200ms).

Procedure

Following set-up for EEG recording, participants were seated in front of a computer monitor, at a viewing distance of 70cm. Phonological and orthographic lexical decision tasks were completed in counterbalanced order. Participants were instructed to respond as quickly and

as accurately as possible to all word stimuli irrespective of cue validity and to avoid blinking or overtly moving their head or eyes. In the ODT, participants were asked to respond yes to words that looked like real words (real words) and no to words that did not (pseudohomophones). In the phonological decision task, participants were asked to respond yes to words that sounded like real words (pseudohomophones) and no to words that did not (nonwords). In order to control for response output processes the study was counterbalanced for response hand (left, right) and response (yes, no) as a between subjects factor. Participants took short breaks between tasks to prevent fatigue. The experimental session lasted approximately two hours (including set up for electrophysiological recording) with most participants completing several different experiments within the session.

Design and Data Analysis

Mean RT and accuracy for 'yes' responses for each task (real words for the orthographic tasks and pseudohomophones for the phonological task) were analysed according to the following design: 2[Group: good decoder, poor decoder] x 2(Task: ODT, PDT) x 2(Cue: valid, invalid) x 2(Visual field: LVF, RVF). The same analysis was used to investigate the effects of experimental manipulations on mean P1 and N1 amplitude and latency for correct 'yes' responses with the inclusion of the following additional factors: 2(Sagittal site: occipital, parietal, temporal) x 2(Hemisphere: LH, RH) or x 2(Laterality: contralateral, ipsilateral). Greenhouse-Geisser corrections were applied where appropriate to control for violations of sphericity. Significant interactions were analysed using break-down ANOVAs for analysis of simple effects. Bonferroni adjusted p-values were used to maintain the family-wise Type 1 error rate and these are reported when the correction changed the significance of the analysis. Due to the small number of subjects, sex was not included as a factor in the initial analyses.

Results

Mean Reaction Time

Mean RT was significantly longer for the phonological ($M=1.00$, $SEM=.023$) in comparison to the orthographic task ($M=.773$, $SEM=.017$), $F(1,24)=172.99$, $MSE=0.016$, $p<.001$, for invalid ($M=.911$, $SEM=.021$) relative to valid trials ($M=.867$, $SEM=.017$), $F(1,24)=26.50$, $MSE=0.004$, $p<.001$, and for LVF ($M=.898$, $SEM=.019$) in comparison to RVF ($M=.879$, $SEM=.019$) trials, $F(1,24)=8.74$, $MSE=0.002$, $p<.001$. Overall mean RT was significantly longer for poor decoders ($M=.947$, $SEM=.026$) in comparison to good decoders ($M=.830$, $SEM=.026$), $F(1,24)=10.01$, $MSE=0.072$, $p<.01$.

There was a significant Task x VF interaction, $F(1,24)=14.53$, $MSE=0.003$, $p<.01$, such that there was a significant RVF advantage for the ODT, $F(1,24)=23.83$, $MSE=0.003$, $p<.001$, but not the PDT ($p>.05$). The Cue x Group interaction approached significance, $F(1,24)=4.02$, $MSE=0.004$, $p=.056$, such that the effect of Cue was significant for good decoders, $F(1,12)=25.19$, $p<.001$, but only approached significance for poor decoders, $F(1,12)=5.02$, $p=.045$ ($p>.05$, Bonferroni corrected), and the effect of Group was significant for both valid, $F(1,24)=14.9$, $MSE=0.032$, $p<.01$, and invalid trials, $F(1,24)=5.97$, $MSE=0.044$, $p<.05$.

Figure 39 shows a trend for a Cue x VF x Group interaction, $F(1,24)=3.95$, $p=.058$, such that the Cue x Group interaction was significant for LVF trials, $F(1,24)=7.34$, $p<.05$. The effect of Cue was significant for good decoders, $F(1,12)=8.96$, $MSE=0.004$, $p<.05$, but not poor decoders ($p>.05$) and the effect of Group was significant for valid trials, $F(1,24)=16.77$, $MSE=0.017$, $p<.001$, but only approached significance for invalid trials, $F(1,24)=3.68$, $MSE=0.024$, $p=.067$. For RVF trials, there were overall effects of Cue, $F(1,24)=39.95$, $MSE=0.003$, $p<.001$, and Group, $F(1,24)=10.18$, $MSE=0.036$, $p<.01$.

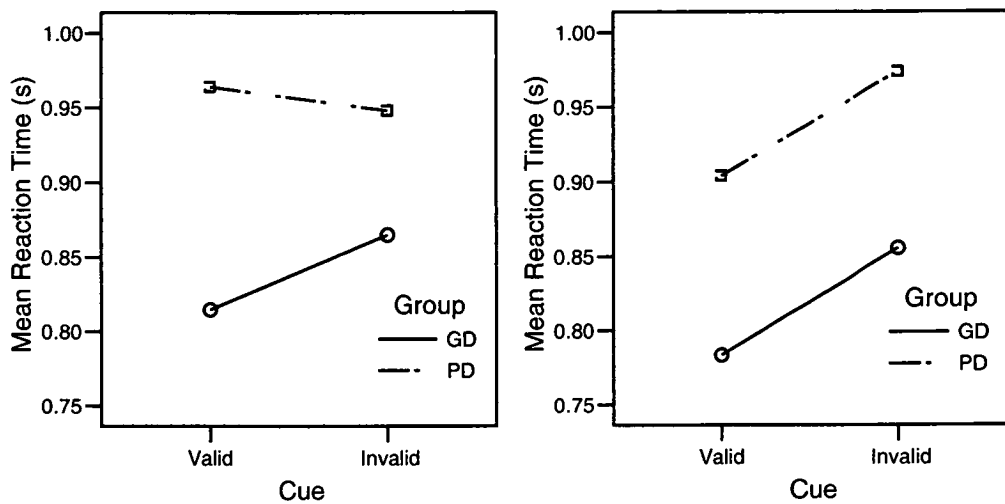


Figure 39. Mean RT for good decoders (GD) and poor decoders (PD) as a function of Cue for LVF (left) and RVF (right) trials.

Figure 40 shows a significant Cue x VF x Group interaction for the phonological task, $F(1,24)=4.82$, $MSE=0.004$, $p<.05$. Good decoders showed a significant effect of Cue, $F(1,12)=8.81$, $MSE=.006$, $p<.05$, and no overall effect of VF ($p>.05$). The Cue x VF interaction was significant for poor decoders, $F(1,12)=12.78$, $MSE=0.006$, $p<.01$, such that RT was shorter for valid in comparison to invalid trials in the RVF, $F(1,12)=8.50$, $MSE=0.006$, $p<.05$, but tended to be shorter for invalid in comparison to valid trials in the LVF, $F(1,24)=4.21$, $MSE=0.006$, $p=.063$. Further, poor decoders tended to show a RVF advantage for valid trials, $F(1,12)=5.67$, $MSE=0.004$, $p<.035$ ($p>.05$, Bonferroni corrected), and a significant LVF advantage for invalid trials, $F(1,12)=14.53$, $MSE=0.004$, $p<.01$. The effect of Group was significant for valid trials overall, $F(1,12)=15.79$, $MSE=0.004$, $p<.01$, and for invalid trials in the RVF, $F(1,12)=6.79$, $MSE=0.004$, $p<.05$, but not the LVF ($p>.05$).

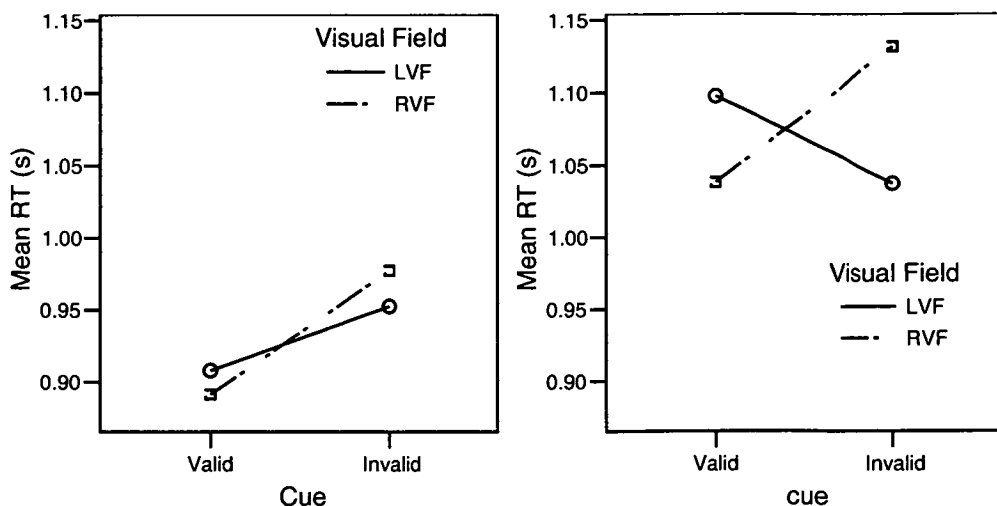


Figure 40. Mean RT for good decoders (left) and poor decoders (right) in response to PHs in the phonological task as a function of Cue and Visual field.

Mean Accuracy

Mean accuracy (%) was significantly greater for the ODT ($M=89.7\%$, $SEM=.782$) in comparison to the PDT ($M=75.6\%$, $SEM=1.50$), $F(1,24)=125.1$, $MSE=138.1$, $p<.001$, and for valid ($M=84.2\%$, $SEM=.939$) in comparison to invalid ($M=81.1\%$, $SEM=1.05$) trials, $F(1,24)=28.3$, $MSE=84.4$, $p<.001$. Overall accuracy was significantly greater for good decoders in comparison to poor decoders, $F(1,24)=16.80$, $MSE=381.3$, $p<.001$. There was a significant VF x Cue interaction, $F(1,24)=16.80$, $MSE=35.1$, $p<.001$, such that there was a significant RVF advantage for valid trials, $F(1,24)=28.45$, $p<.001$, and a trend for a LVF advantage for invalid trials, $F(1,24)=4.32$, $MSE=55.8$, $p=.048$ ($p>.05$, Bonferroni corrected). However, the effect of Cue was significant for both LVF, $F(1,24)=4.64$, $p<.05$, and RVF trials, $F(1,24)=49.07$, $p<.001$. The Task x Cue interaction was also significant, $F(1,24)=16.12$, $MSE=80.9$, $p<.01$, indicating that the effect of Cue was significant for the PDT, $F(1,24)=36.97$, $MSE=97.6$, $p<.001$, but not the ODT ($p>.05$). There was a significant Task x Group interaction, $F(1,24)=13.42$, $MSE=138.1$, $p<.05$, such that the main effect of Group was greater for the PDT, $F(1,24)=17.82$, $MSE=425.1$, $p<.001$, than the ODT, $F(1,24)=7.25$, $MSE=94.3$, $p<.05$.

Electrophysiological Data

Figures 41-44 show grand mean averaged waveforms for each task and visual field as a function of Cue and Group. In terms of the early P1 and N1 components, the largest differences between good and poor phonological decoders appears to be at LH parietal sites (P3) such that poor phonological decoders show greater N1 amplitude relative to good decoders, particularly for RVF for both the orthographic (Figure 42) and phonological (Figure 44) tasks. In contrast, for LVF trials good decoders appear to show greater N1 amplitude at RH parietal (P4) and temporal (P8) sites, particularly for the orthographic task (Figure 41) and particularly for valid trials.

P1 Amplitude

P1 was significantly greater at parietal ($M=4.99$, $SEM=.458$) relative to occipital ($M=3.99$, $SEM=.279$) and temporal ($M=3.72$, $SEM=.262$) sites, $F(2,48)=8.17$, $MSE=35.6$, $p<.01$, at ipsilateral ($M=4.94$, $SEM=.309$) relative to contralateral ($M=3.52$, $SEM=.289$) sites, $F(1,24)=73.91$, $MSE=8.6$, $p<.001$, at RH ($M=4.72$, $SEM=.320$) relative to LH sites ($M=3.74$, $SEM=.385$), $F(1,24)=5.52$, $MSE=53.6$, $p<.05$, and for RVF ($M=4.67$, $SEM=.342$) in comparison to LVF trials ($M=3.79$, $SEM=.366$), $F(1,24)=4.52$, $MSE=53.3$, $p<.05$. However, these effects were modified by a significant Laterality x VF interaction, $F(1,24)=5.52$, $MSE=53.6$, $p<.05$, such that P1 amplitude at ipsilateral sites was significantly greater for RVF (RH) in comparison to LVF (LH) trials, $F(1,24)=7.09$, $MSE=65.2$, $p<.05$.

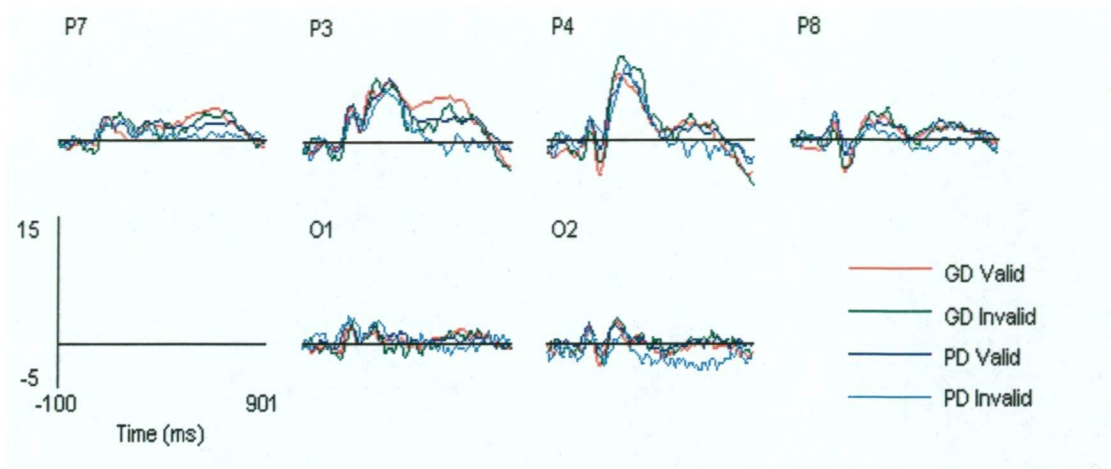


Figure 41. Grand mean averaged waveforms for LVF trials in the ODT as a function of Cue (valid vs. invalid) for good decoders (GD) and poor decoders (PD).

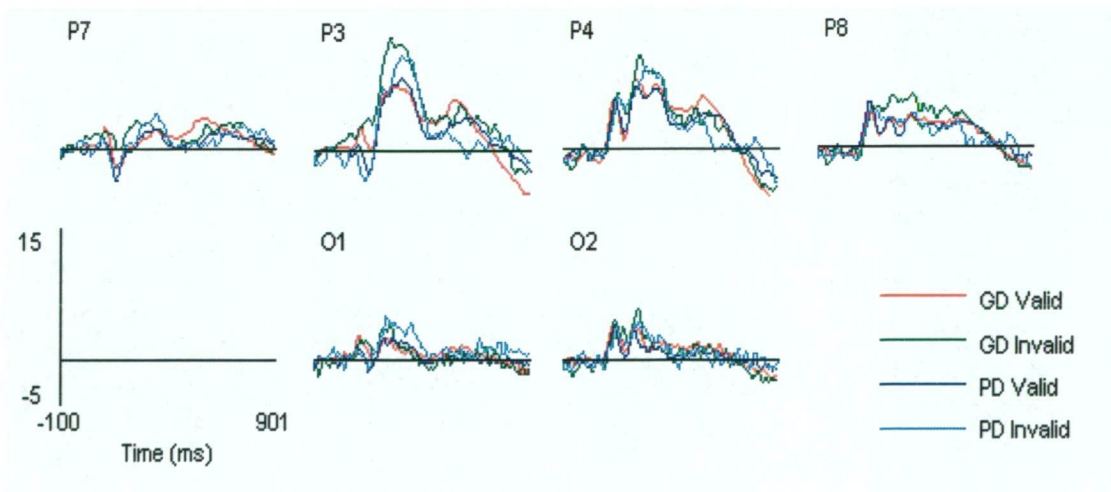


Figure 42. Grand mean averaged waveforms for the RVF trials in the ODT as a function of Cue (valid vs. invalid) for good decoders (GD) and poor decoders (PD).

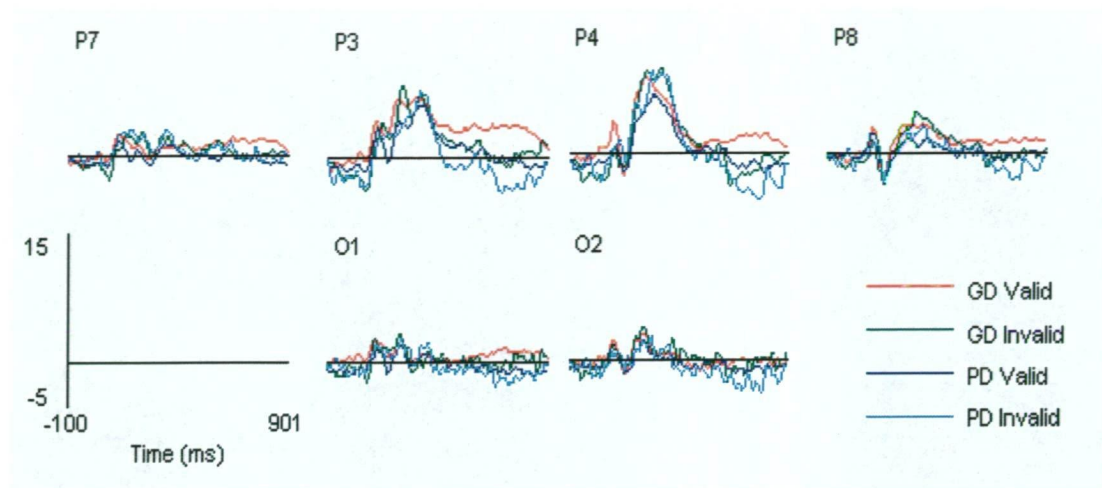


Figure 43. Grand mean averaged waveforms for the LVF trials in the PDT as a function of Cue (valid vs. invalid) for good decoders (GD) and poor decoders (PD).

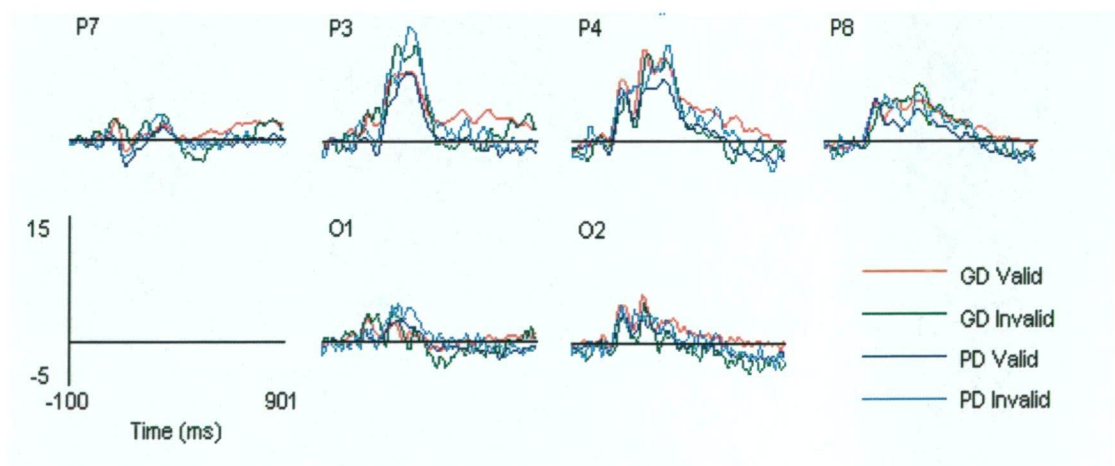


Figure 44. Grand mean averaged waveforms for the RVF trials in the PDT as a function of Cue (valid vs. invalid) for good decoders (GD) and poor decoders (PD).

At occipital sites there was a significant main effect of Cue, $F(1,24)=8.77$, $MSE=4.74$, $p<.01$, indicating greater P1 amplitude for invalid in comparison to valid trials. There was also a trend for a Cue x Group interaction at occipital sites, $F(1,24)=3.78$, $MSE=4.74$, $p=.064$, such that poor decoders showed significantly greater P1 amplitude for invalid in comparison to valid trials, $F(1,12)=11.33$, $MSE=5.04$, $p<.01$, whereas the effect of Cue was not significant for good decoders ($p>.05$), and there were no significant between group differences ($ps>.05$).

Figure 45 shows a trend for a Cue x Laterality x Group interaction at occipital sites, $F(1,24)=3.60$, $MSE=1.47$, $p=.07$. Good decoders showed a significant Cue x Laterality interaction, $F(1,12)=5.67$, $MSE=1.07$, $p<.05$, such that P1 amplitude tended to be greater for invalid in comparison to valid trials at contralateral sites, $F(1,12)=3.39$, $MSE=2.14$, $p=.091$, whereas poor decoders showed significantly greater P1 amplitude for invalid in comparison to valid trials regardless of hemisphere, $F(1,12)=11.33$, $MSE=5.04$, $p<.01$. The Laterality x Group interaction for invalid trials was significant, $F(1,24)=4.69$, $MSE=0.695$, $p<.05$. Poor decoders showed significantly greater P1 amplitude at ipsilateral in comparison to contralateral sites, $F(1,12)=26.43$, $MSE=0.568$, $p<.001$, whereas good decoders showed no laterality differences ($p>.05$). For valid trials, P1 amplitude was greater at ipsilateral in comparison to contralateral sites regardless of Group, $F(1,24)=68.47.24$, $MSE=0.257$, $p<.001$. There were no significant between group differences ($ps>.05$).

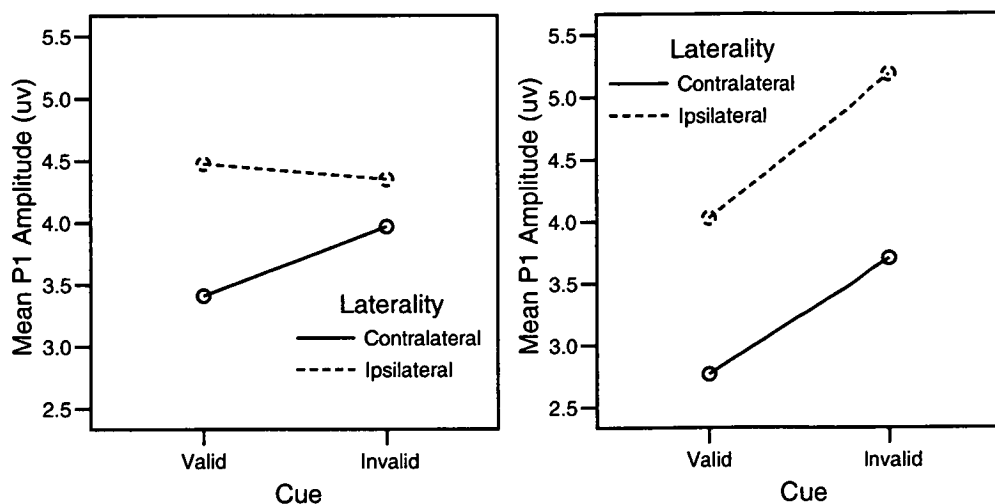


Figure 45. Mean P1 amplitude for valid and invalid trials at ipsilateral and contralateral occipital sites for good decoders (left) and poor decoders (right).

Figure 46 shows a significant Cue x Hemisphere x Group interaction at parietal sites, $F(1,24)=8.51$, $MSE=1.06$, $p<.01$. Good decoders showed a significant Hemisphere x Cue interaction, $F(1,12)=7.68$, $MSE=0.723$, $p<.05$, such that P1 amplitude tended to be greater for invalid than valid trials in the LH and greater for valid in comparison to invalid trials in the RH, however, these differences were not statistically significant ($ps>.05$). Poor decoders showed significantly greater P1 amplitude in the RH in comparison to the LH overall, $F(1,12)=16.35$, $MSE=3.35$, $p<.01$. There was a significant Cue x Group interaction in the RH, $F(1,24)=4.42$, $MSE=2.64$, $p<.05$, such that poor decoders showed greater amplitude for invalid in comparison to valid trials and good decoders showed greater amplitude for valid in comparison to invalid trials, however, these differences were not statistically significant ($ps>.05$).

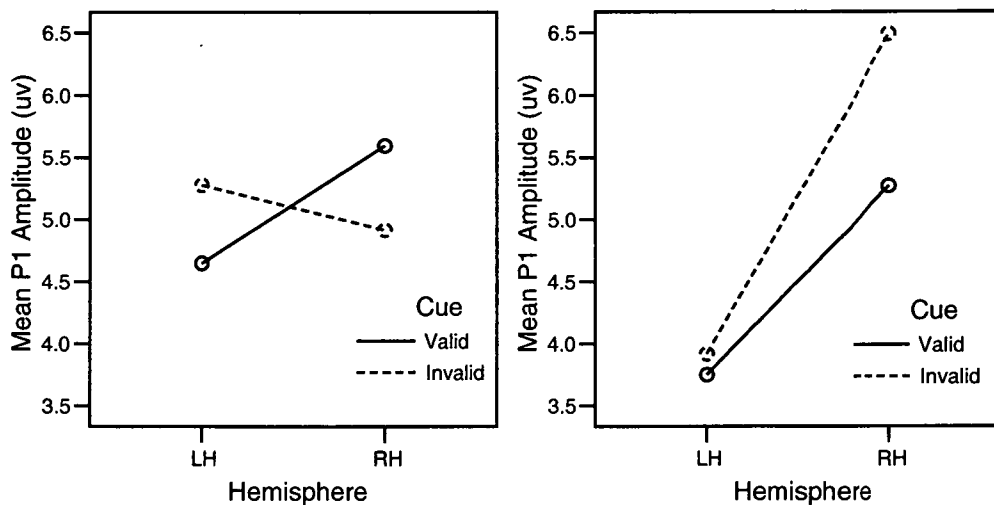


Figure 46. Mean P1 amplitude as a function of Cue and Hemisphere for good decoders (left) and poor decoders (right).

P1 Latency

P1 latency was significantly shorter at contralateral ($M=113.3$, $SEM=1.82$) in comparison to ipsilateral sites ($M=131.3$, $SEM=1.27$), $F(1,24)=120.0$, $MSE=839.9$, $p<.001$. There was a significant Laterality x Cue interaction, $F(1,24)=4.51$, $MSE=542.2$, $p<.05$, such that the effect of Cue was significant at contralateral sites, $F(1,24)=6.50$, $MSE=722.2$, $p<.05$, indicating shorter P1 latency for valid ($M=110.7$, $SEM=2.19$) relative to invalid ($M=116.2$, $SEM=2.07$) trials. There was a significant Cue x Sagittal site interaction, $F(2,48)=3.87$, $MSE=241.9$, $p<.05$, such that P1 latency was shorter for valid in comparison to invalid trials at occipital sites, $F(1,24)=4.96$, $MSE=352.4$, $p<.05$. This effect only approached significance at parietal sites, $F(1,24)=3.20$, $MSE=511.9$, $p=.086$, and was non-significant at temporal sites ($p>.05$).

The Cue x Group interaction approached significance, $F(1,24)=3.40$, $MSE=648.0$, $p=.077$, such that the main effect of Cue was significant for good decoders, $F(1,12)=7.14$, $MSE=545.6$, $p<.05$, and not for poor decoders ($p>.05$). Good decoders showed greater P1 latency for invalid in comparison to valid trials (126ms vs. 121ms), and this tended to be greater in

comparison to poor decoders, $F(1,24)=3.59$, $MSE=107.3$, $p=.07$. Figure 47 shows a trend for a VF x Cue x Group interaction, $F(1,24)=3.87$, $MSE=26.1$, $p=.061$. There was a significant effect of Cue for LVF trials, $F(1,24)=6.14$, $MSE=30.6$, $p<.05$, and a significant Cue x Group interaction for RVF trials, $F(1,24)=5.63$, $MSE=49.5$, $p<.05$, such that the effect of Cue approached significance for good decoders, $F(1,12)=5.01$, $p=.045$ ($p>.05$, Bonferroni corrected), but not for poor decoders ($p>.05$). There was a significant VF x Group interaction for invalid trials, $F(1,24)=6.38$, $MSE=62.5$, $p<.05$. Poor decoders showed significantly shorter P1 latency for RVF invalid trials in comparison to good decoders, $F(1,24)=8.21$, $MSE=95.48$, $p<.01$, and this tended to be shorter in comparison to LVF trials ($p=.084$). The effect of Cue was not significant for poor decoders in either VF ($ps>.05$).

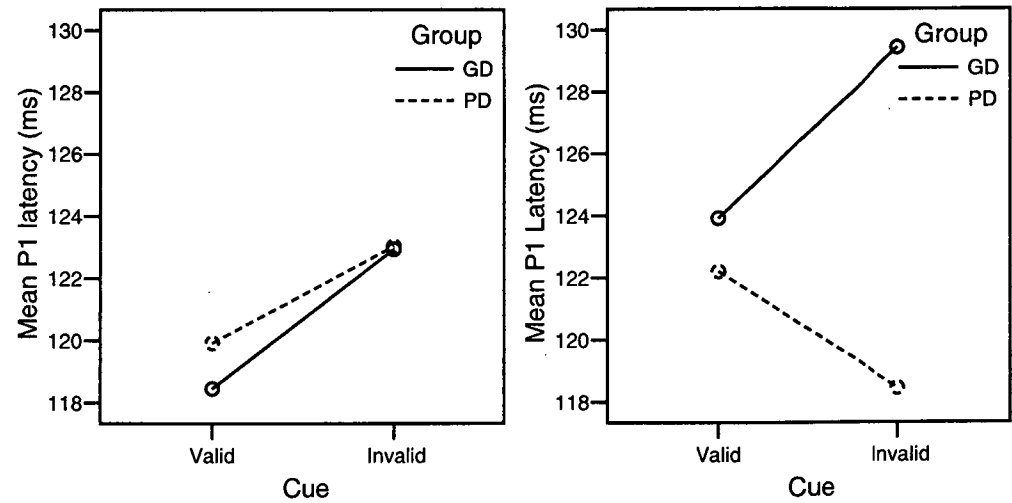


Figure 47. Mean P1 latency as a function of Group and Cue for LVF (left) and RVF (right) trials.

The VF x Laterality x Group interaction was significant at parietal sites, $F(1,24)=6.82$, $MSE=341.8$, $p<.05$ (see Figure 48). Breakdown analyses revealed a significant VF x Group interaction at contralateral parietal sites, $F(1,24)=7.45$, $MSE=592.4$, $p<.05$, such that poor decoders tended to show shorter latency for RVF (LH) in comparison to LVF (RH) trials, $F(1,12)=5.55$, $MSE=409.2$, $p=.036$ ($p>.05$ Bonferroni corrected), and this tended to be shorter in

comparison to good decoders, $F(1,24)=5.27$, $MSE=303.9$, $p=.031$ ($p>.05$ Bonferroni corrected) who showed no VF differences ($p>.05$).

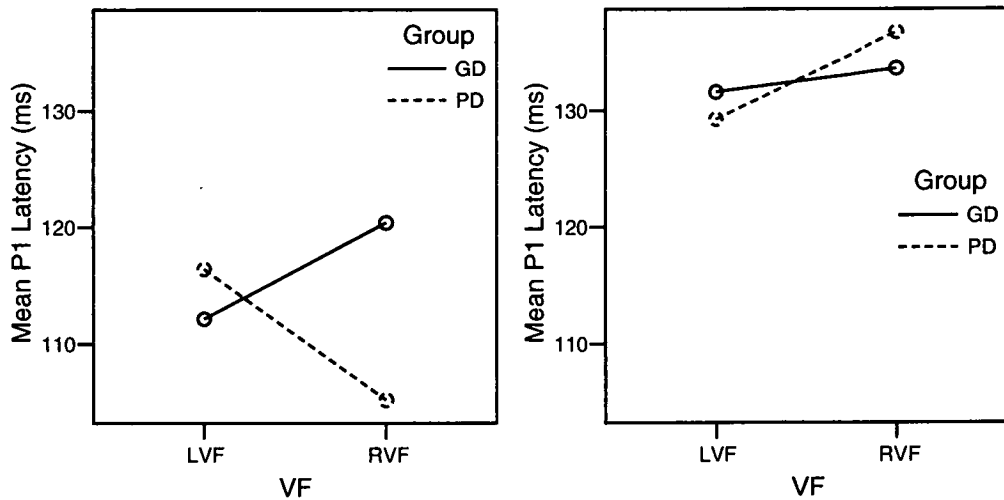


Figure 48. Mean P1 latency for good decoders (GD) and poor decoders as a function of VF at contralateral (left) and ipsilateral (right) parietal sites.

N1 Amplitude

There was a significant main effect of Laterality, $F(1,24)=67.44$, $MSE=36.3$, $p<.001$, indicating significantly greater N1 amplitude at contralateral ($M=-2.57$, $SEM=.441$) in comparison to ipsilateral ($M=.228$, $SEM=.345$) sites. The VF main effect was significant, $F(1,24)=8.25$, $MSE=36.3$, $p<.01$, indicating significantly greater N1 amplitude for LVF ($M=-1.96$, $SEM=.495$) in comparison to RVF ($M=-.390$, $SEM=.397$) trials. However, this was modified by a significant VF x Sagittal interaction, $F(2,48)=4.78$, $p<.05$, such that the effect of VF was significant at parietal, $F(1,24)=11.45$, $p<.01$, and occipital sites, $F(1,24)=8.30$, $MSE=4.72$, $p<.01$, and only approached significance at temporal sites ($p=.07$).

The Hemisphere x Group interaction was significant at parietal sites, $F(1,24)=8.11$, $MSE=23.2$, $p<.01$. Good decoders tended to show greater N1 amplitude in the RH in comparison to the LH, $F(1,12)=3.79$, $MSE=31.2$, $p=.075$, whereas poor decoders tended to show greater N1 amplitude in the LH in comparison to the RH overall, $F(1,12)=4.79$, $MSE=15.2$, $p=.049$ ($p>.05$,

Bonferroni corrected) and this tended to be greater in comparison to good decoders, $F(1,24)=3.47$, $MSE=9.6$, $p=.075$. However, this interaction was qualified further by a significant VF x Laterality x Group interaction at parietal sites (see Figure 49), $F(1,24)=8.11$, $MSE=23.2$, $p<.01$. The VF x Group interaction was significant at contralateral sites, $F(1,24)=5.32$, $MSE=9.26$, $p<.05$, such that good decoders showed significantly greater N1 amplitude for LVF (RH) in comparison to RVF (LH) trials, $F(1,24)=8.73$, $MSE=12.6$, $p<.05$, whereas poor decoders did not show any VF differences and there were no significant between group differences ($ps>.05$). There was a significant Laterality x Group interaction for RVF trials, $F(1,24)=4.77$, $MSE=7.14$, $p<.05$, such that poor decoders showed greater N1 amplitude at contralateral (LH) in comparison to ipsilateral (RH) sites, $F(1,12)=13.24$, $MSE=8.57$, $p<.01$, and this tended to be greater in comparison to good decoders, $F(1,24)=4.02$, $MSE=15.9$, $p=.056$. Good decoders did not show any laterality differences for RVF trials.

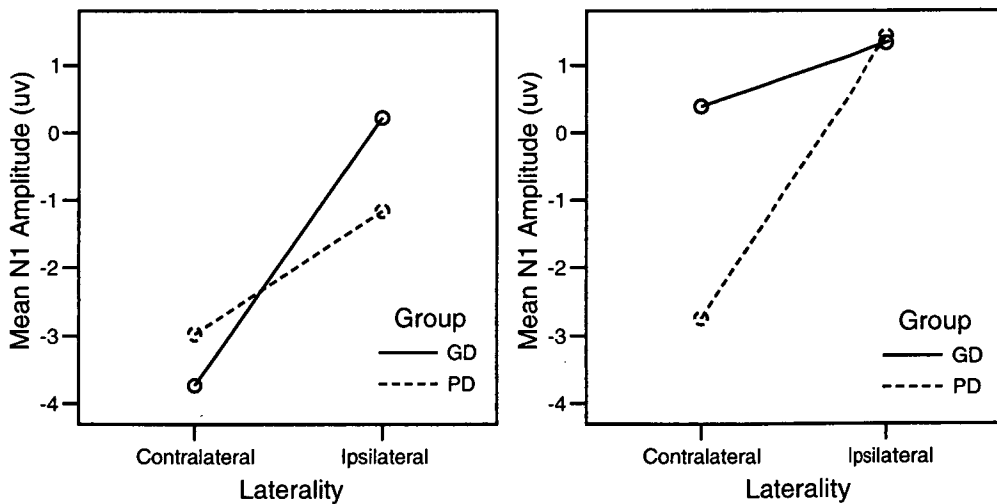


Figure 49. Mean N1 amplitude of good decoders (GD) and poor decoders (PD) at contralateral and ipsilateral sites for LVF (left) and RVF (right) trials.

For the ODT, there was a significant Laterality x Cue x Group interaction at occipital sites, $F(1,24)=8.00$, $MSE=1.96$, $p<.01$. The Laterality x Cue interaction was significant for poor decoders, $F(1,12)=7.11$, $MSE=2.85$, $p<.05$, such that N1 amplitude tended to be greater for

invalid in comparison to valid trials at contralateral sites, $F(1,12)=4.51$, $MSE=3.66$, $p=.055$. There were no significant Group differences ($ps>.05$). However, this interaction was qualified further by a significant Cue x VF x Hemisphere x Group interaction for the ODT at occipital sites, $F(1,24)=8.00$, $MSE=1.96$, $p<.01$. The Cue x Hemisphere x Group interaction was significant for LVF trials (see Figure 50), $F(1,24)=6.49$, $MSE=6.49$, $p<.05$. Good decoders showed significantly greater N1 amplitude in the RH than the LH for LVF trials overall, $F(1,12)=16.06$, $MSE=3.35$, $p<.01$. However poor decoders showed a significant Cue x Hemisphere interaction, $F(1,12)=10.14$, $MSE=1.58$, $p<.01$, indicating significantly greater N1 amplitude in the RH than the LH for LVF invalid trials, $F(1,12)=12.12$, $MSE=6.88$, $p<.01$, with only a trend observed for LVF valid trials, $F(1,12)=4.09$, $MSE=2.96$, $p=.066$. The effect of Cue was non-significant for both groups ($ps>.05$). For RVF trials, N1 amplitude was significantly greater in the LH in comparison to the RH overall, $F(1,24)=14.65$, $MSE=3.95$, $p<.01$, and there were no significant between group differences or significant effects of Cue ($ps>.05$).

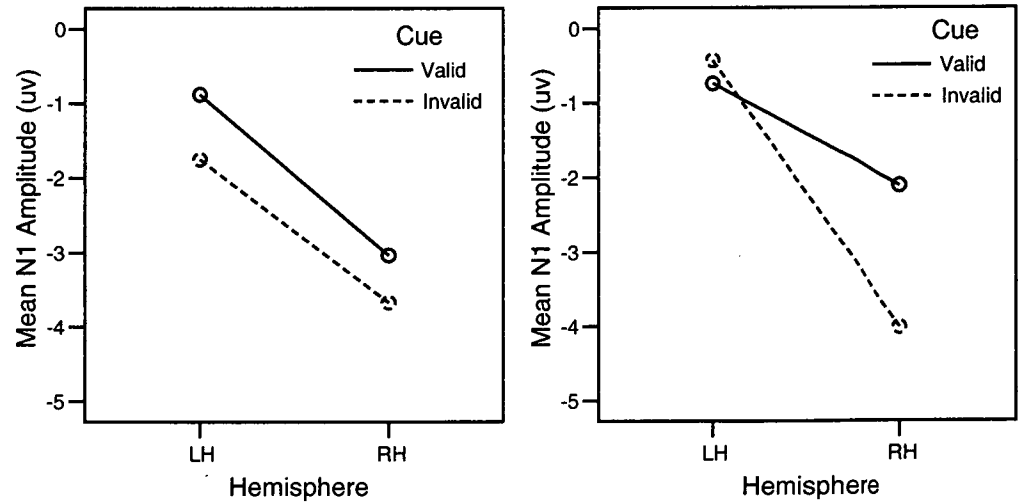


Figure 50. Mean N1 amplitude at occipital sites for LVF trials on the ODT as a function of Cue and Hemisphere for good decoders (left) and poor decoders (right).

N1 Latency

N1 latency was significantly shorter at contralateral ($M=168.6$, $SEM=1.52$) in comparison to ipsilateral sites ($M=178.31$, $SEM=1.40$), $F(1,24)=47.90$, $MSE=608.4$, $p<.001$, and at parietal ($M=170.8$, $SEM=1.50$), in comparison to temporal ($M=173.8$, $SEM=1.55$), and occipital ($M=175.9$, $SEM=1.33$) sites, $F(2,48)=8.90$, $MSE=383.2$, $p<.01$. The Laterality x Cue interaction was significant, $F(1,24)=14.45$, $MSE=269.1$, $p<.01$, such that N1 latency was shorter for valid in comparison to invalid trials at contralateral sites (167ms vs. 172ms), $F(1,24)=8.77$, $MSE=405.7$, $p<.01$, and tended to be shorter for invalid in comparison to valid trials at ipsilateral sites (177ms vs. 180ms), $F(1,24)=4.10$, $MSE=444.5$, $p=.054$.

Figure 51 shows a significant Task x Cue x Group interaction at contralateral sites, $F(1,24)=4.60$, $MSE=276.1$, $p<.05$. There was a significant Cue x Group interaction at contralateral sites for the PDT, $F(1,24)=4.54$, $MSE=57.8$, $p<.05$. The effect of Cue was significant for good decoders, $F(1,12)=11.45$, $MSE=220.1$, $p<.01$, indicating significantly shorter N1 latency for valid in comparison to invalid trials. However, the effect of Cue was non-significant for poor decoders and there were no overall between group differences ($ps>.05$). At ipsilateral sites for the PDT, N1 latency tended to be shorter for invalid in comparison to valid trials (176ms vs. 181ms) regardless of Group, $F(1,24)=3.28$, $MSE=438.0$, $p=.083$. For the ODT, N1 latency tended to be shorter for valid in comparison to invalid trials at contralateral sites, regardless of Group, $F(1,24)=4.21$, $MSE=276.1$, $p=.051$, and there were no significant effects of Cue or Group at ipsilateral sites ($ps>.05$).

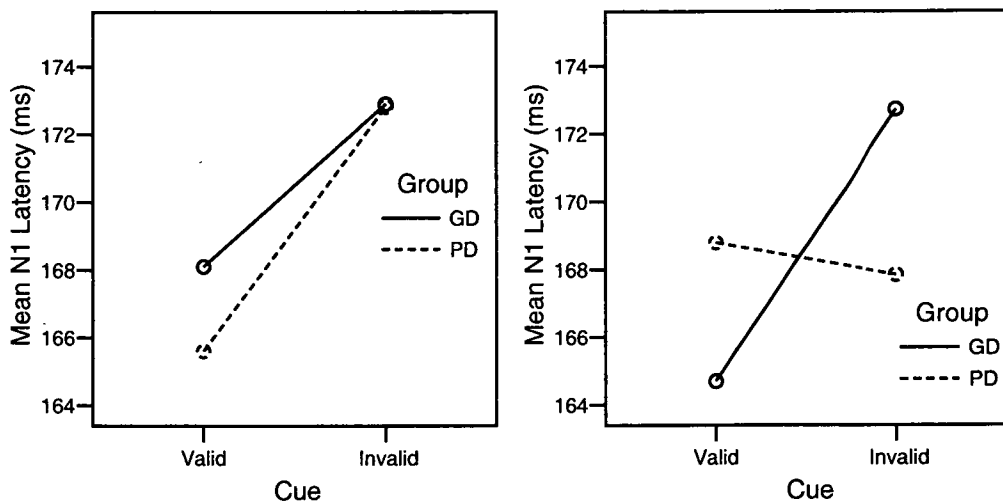


Figure 51. Mean N1 latency for good decoders (GD) and poor decoders (PD) at contralateral sites as a function of Cue for the ODT (left) and PDT (right).

Figure 52 shows a trend for a Laterality x VF x Group interaction, $F(1,24)=3.18$, $MSE=540.2$, $p=.087$. The VF x Group interaction was significant at contralateral sites, $F(1,24)=13.98$, $MSE=529.0$, $p<.01$. Good decoders showed shorter N1 latency for LVF ($M=166.3$, $SEM=1.94$) in comparison to RVF ($M=172.9$, $SEM=2.41$) trials, $F(1,12)=7.47$, $MSE=452.5$, $p<.05$. Poor decoders showed shorter latency for RVF ($M=165.2$, $SEM=2.01$) in comparison to LVF trials ($M=172.4$, $SEM=3.57$), $F(1,12)=6.65$, $MSE=605.4$, $p<.05$, and this was significantly shorter in comparison to good decoders, $F(1,24)=8.60$, $MSE=56.1$, $p<.01$.

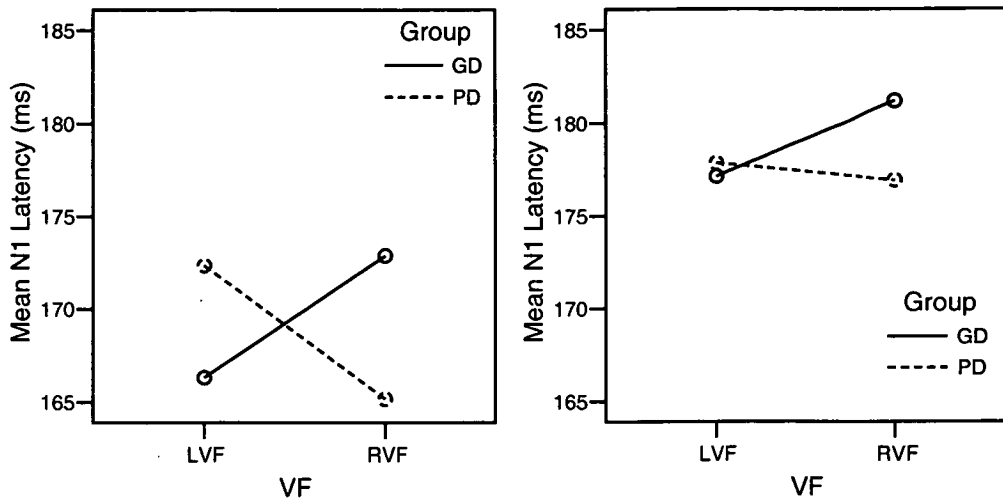


Figure 52. Mean N1 latency for good decoders (GD) and poor decoders (PD) as a function of VF at contralateral (left) and ipsilateral (right) sites.

Discussion

As expected, lexical decision times were longer and accuracy was lower for the phonological in comparison to the orthographic task (Olson et al., 1985; Stanovich & Siegel, 1994). This lexicality effect is consistent with previous research and reflects the processing benefit of accessing existing orthographic representations in the orthographic task and the greater demands on resources and/or slower speed of processing due to the sequential nature of processing in the phonological task (Breznitz, 2003). There was a significant RVF decision time advantage for the orthographic task. This is consistent with previous research findings (Hardyck et al., 1985; Ortells et al., 1998) and with the proposal that the LH and RH use different strategies that are specialised for parallel lexical and serial sublexical processing respectively (Chiarello, 1988; Lindell & Nicholls, 2003). Poor decoders showed significantly longer decision times and lower accuracy overall in comparison to good decoders and a reduction in accuracy for the phonological task overall. These findings are consistent with the selection of groups on the basis of phonological decoding ability and with previous research reporting greater differences

between dyslexics and controls on phonological relative to orthographic decision tasks (Breznitz, 2003; Olson et al., 1985; Stanovich & Siegel, 1994).

As predicted, decision times were shorter and accuracy greater for words preceded by valid in comparison to invalid spatial cues (McCann et al., 1992; Ortells et al., 1998). These findings are consistent with early selection accounts of word recognition (Treisman, 1988; Treisman & Gelade, 1980), suggesting that spatial attention affects word recognition prior to lexical access. However, consistent with familiarity sensitive models of attention in reading (LaBerge & Brown, 1989; Mozer & Behrmann, 1990) and similar dual-route conceptualisations (Cestnick & Coltheart, 1999) the effect of spatial cueing on accuracy was greater for the phonological in comparison to the orthographic task.

Poor decoders showed longer RT to valid trials in comparison to good decoders, suggesting less benefit of early spatial selection. Whereas good decoders showed a significant spatial cuing effect on RT in both visual fields, the spatial cueing effect was non significant for poor decoders for words presented in the LVF. This effect was particularly pronounced for the phonological task. A disruption of spatial cuing in the LVF is consistent with previous research findings of LVF inattention or mini-neglect in dyslexic populations as well as the findings of Experiment 4 in the present series of experiments and may be related to functioning of RH posterior parietal areas (Eden et al., 2003; Facoetti & Turatto, 2000; Hari et al., 2001). It is known from neuroimaging research that the parietal cortex is involved in the shifting of spatial attention (Corbetta, Kincade, Ollinger, McAvoy, & Shulman, 2000; Corbetta et al., 1993). Unilateral spatial neglect as a consequence of RH parietal damage can result in reduced ability to report the left side of pages, lines, or words during reading. Patients with right posterior lesions are able to identify words correctly but fail to report letters on the contralesional (left) side of nonwords (Brunn & Farah, 1991; Sieroff et al., 1988). The fact that group differences in the effects of spatial attention were greater for LVF trials during phonological decoding is consistent with difficulty in the sequential allocation of spatial attention particularly when presented

initially to the contralateral RH that is specialised for sublexical processing (e.g., Chiarello, 1988; Ellis, 2004).

According to Vidyasagar (1999, 2001, 2004) the spatial attention difficulties observed in dyslexia are as associated with a deficiency in a magnocellular mediated attentional spotlighting mechanism that originates in the PPC and selects locations for subsequent identification in extra-striate areas of the ventral system. Attentional modulation of the P1 and N1 components is thought to index this spotlighting process (Eimer, 1998; Mangun, 1995). However, response relevance of unattended locations and the relative discriminability of stimulus attributes influences attentional modulation of these components, despite evidence for consistent reaction time effects, suggesting that attentional modulation is not necessarily obligatory (Eimer, 1998). In the present study attentional modulation of P1 and N1 amplitude was not consistently observed. It is possible that the processing demands of performing lexical decisions are different to (or mask) those required to perform simple detection or discrimination tasks that are usually used in covert orienting paradigms. There was however some evidence for an interaction in the predicted direction for P1 amplitude at RH parietal sites, such that good decoders showed greater P1 amplitude for valid in comparison to invalid trials, whereas poor decoders showed greater P1 amplitude for invalid in comparison to valid trials. Although these effects did not reach statistical significance and should be interpreted with caution, they are consistent with the RH deficit hypothesis of dyslexia.

There were also differences in the lateralization of N1 amplitude at parietal sites, such that good decoders tended to show greater N1 amplitude in the RH than the LH, whereas poor decoders tended to show greater N1 amplitude in the LH than the RH, and this tended to be greater in comparison to good decoders. At contralateral parietal sites, poor decoders showed greater N1 amplitude for RVF trials in the LH than the RH and this tended to be greater in comparison to good decoders who showed greater N1 amplitude for LVF (RH) relative to RVF (LH) trials. Similarly, at contralateral sites, good decoders showed shorter N1 latency overall for

LVF than RVF trials, whereas poor decoders showed shorter latency overall for RVF in comparison to LVF trials and in comparison to good decoders. The greater N1 amplitude and reduction in N1 latency observed in the LH for poor decoders may be a compensatory mechanism that has developed due to a RH deficit and may also indicate reliance on the whole word recognition strategy of the LH. For example, it has been suggested that the LH uses a parallel strategy whereas the RH uses a serial strategy that are specialised for whole word recognition and sequential sublexical processing respectively (Chiarello, 1988; Lindell & Nicholls, 2003). McPherson et al. (1996) found that dysphonetics showed greater N1 amplitude in the left hemisphere during a phonological priming task. This was accompanied by few behavioural differences suggesting that the difference occurred due to compensatory processing.

For the orthographic decision task, poor decoders tended to show greater N1 amplitude for invalid in comparison to valid trials at contralateral occipital sites. However, this effect was largely due to a reduction in N1 amplitude in the RH for validly cued targets presented in the LVF. This finding is consistent with the differences observed for LVF trials in the behavioural data and with the hypothesis that differences in processing within the RH are associated with reading difficulty. Wimmer et al. (2002) also found that dyslexics showed a reduction in N1 amplitude at RH sites for words but not pseudo-words during a word naming task.

Consistent with some previous research findings (Anllo-Vento & Hillyard, 1995; Wascher & Tipper, 2004), P1 and N1 latency were modulated by attention in the present study such that shorter latency was observed for valid in comparison to invalid trials. The effect of spatial attention on P1 latency was found to be significant for good decoders and not for poor decoders, particularly for RVF trials. N1 latency at contralateral sites was also significantly shorter for valid in comparison to invalid trials, particularly at parietal sites. However, for the phonological task, good decoders but not poor decoders showed attentional modulation of N1 latency at contralateral sites. Together these findings suggest that valid cues did not afford the same processing gain in terms of speeding up neural processing for poor decoders as they did for

good decoders. As well as a reduction in P1 latency for RVF invalid trials, poor decoders also showed an overall reduction in P1 latency at LH parietal sites for words presented in the RVF overall. These findings suggest that poor decoders were faster to engage the LH for processing the RVF even when attention was initially directed to the LVF. These findings are consistent with the proposal that dyslexics are more distracted by visual stimuli presented in the RVF (Facoetti & Turatto, 2000; Hari et al., 2001). According to hemispheric models of attentional control (Kinsbourne, 1970, 2003), hyperactivity of the LH could be due to a RH deficit in attentional control.

Poor decoders showed a trend for a reversal of the spatial cuing effect for the phonological task, such that RT tended to be greater for valid in comparison to invalid trials. Although preliminary, these findings could indicate inhibition of return or a bias against returning attention to previously attended locations under these conditions. However, inhibition of return is usually observed with peripheral rather than central cues. Chasteen and Pratt (1999) investigated inhibition of return in a peripheral cueing experiment involving the discrimination of low and high frequency words and nonwords. Inhibition of return was found to be greater for low relative to high frequency words. It was suggested that inhibition is greater for items such as low frequency words which require the reorienting of attention to several locations of the cued area. Although mid brain structures such as the superior colliculus are thought to be involved in Inhibition of return, posterior parietal damage has also been found to disturb inhibition of return (Vivas, Humphreys, & Fuentes, 2003).

The presence of inhibition of return for poor decoders in the present data may relate to the fact that poor decoders also showed a significant effect of cue in the non-predicted direction at ipsilateral occipital sites such that P1 amplitude was significantly greater for invalid relative to valid trials. A similar reversal in the P1 attention effect has been found in some other studies that have used both informative and uninformative peripheral cues at long SOAs (Doallo et al., 2004; Eimer, 1994; Hopfinger & Mangun, 1998). It has been argued that the reversal in the P1 effect

reflects sensory inhibition of sensory processing for same location targets (Doallo et al., 2004; Eimer, 1994) and the interaction between voluntary orienting and an inhibitory effect, such that the inhibitory effect overcomes the voluntary process at longer SOAs. It is possible that poor decoders were less efficient at inhibiting or disengaging attentional processing in the hemisphere contralateral to the cued location on invalid trials as reflected by increased P1 amplitude at ipsilateral sites.

Whereas the present study found spatial cuing effects for RVF but not LVF trials among adult poor phonological decoders, Facoetti et al. (2001) found a greater spatial cuing effect in the LVF than the RVF among dyslexic children. Although these findings appear conflicting, they were both due to differences in the LVF with longer decision times found for valid trials in the present study and for invalid trials in the Facoetti et al. study. The discrepancy in these findings could be due to task-related requirements or the development of compensation strategies in the high functioning adult sample recruited in the present study.

Considering the evidence for sex differences in the hemispheric lateralisation of both phonological processing and spatial attention (Gur et al., 2000; Shaywitz et al., 1995), as well as the sex differences observed in the present series of experiments, it is possible that sex differences contributed to the findings of the present study. Although the small number of male subjects in the present experiment prevented preliminary investigation of sex differences, future research investigating sex differences using similar paradigms is warranted. Another possible limitation of the present study is that despite instruction to keep their eyes on the centre of the screen, it is possible that participants had enough time to make saccades toward the cue location. Unfortunately it was not possible to use short durations in the present study due to the difficulty of the lexical decision tasks, particularly for poor readers.

In summary, the good decoders in the present study showed consistent behavioural effects of spatial attention on word recognition which supports early selection models of word recognition. However, poor phonological decoders show less benefit of spatial attention

particularly for words presented in the LVF when processing was biased towards phonological processing and therefore required the left to right serial allocation of attention to individual letters within a word. These behavioural differences were accompanied by an absence of the attentional modulation of either P1 or N1 latency for poor decoders and the lack of N1 latency modulation was greatest for the phonological task. There was an overall reversal of the lateralisation of the N1 component. Good decoders showed greater N1 amplitude at RH parietal sites and poor decoders showed greater N1 amplitude at LH parietal sites particularly for words presented in the contralateral visual field and a similar reversal was found for N1 latency. Further, poor decoders showed a reduction in N1 amplitude at RH occipital sites for LVF valid trials. There was also some preliminary evidence for a lack of attentional modulation of P1 amplitude at RH parietal sites. Together these findings are generally consistent with previous research findings which have indicated that dyslexia is associated with a spatial attention characterised by LVF inattention due to a RH posterior parietal impairment. In the present study, there was evidence that poor phonological decoders compensated for processing differences in the RH through reliance on and/or less inhibition of attentional processing in the LH. Whereas it is possible that sex differences contributed to some of the present findings, further research is required to investigate this possibility.

CHAPTER 11 - GENERAL DISCUSSION AND CONCLUSIONS

The present series of experiments were conducted to investigate behavioural and electrophysiological indices of attentional processes in good and poor phonological decoders. The findings of Experiment 1 indicated that poor phonological decoders were slower to search for conjunction of features in comparison to good phonological decoders, particularly when defined by the features of form and motion. These findings were consistent with differences in the guidance of spatial attention, but it was not possible to determine whether this was due to differential excitation of relevant locations (Wolfe et al., 1989), or inhibition of irrelevant locations (Treisman & Sato, 1990), a combination of both, or some other mechanism. Experiments 2-5 aimed to examine attentional processes among good and poor phonological decoders in more detail by examining the specific mechanisms involved in spatial attention and their relationship to components of the ERP waveform (e.g., N1, P1, N2) that are thought to index early attentional and perceptual processes.

The results of Experiment 2 indicated that the behavioural effects of cue-size and flanker manipulations were different for good and poor phonological decoders. Poor decoders showed a greater RT cost for incompatible stimuli preceded by large cues indicating greater difficulty in focussing the spatial scale of attention and suppressing information at unattended locations. This is consistent with the suggestion that the inhibition of distractors is important for successful reading (LaBerge & Brown, 1989) and developing grapheme-phoneme conversion strategies (Brosnan et al., 2002). Under these conditions good decoders showed greater N1 amplitude for incompatible stimuli in the RH, and this effect only approached significance in the LH for poor decoders. Further, whereas good decoders showed bilateral attentional modulation as a function of cue-size, this was only significant in the RH for poor decoders. These findings indicate hemispheric differences in terms of RH involvement in focussing and LH involvement in orienting or cue-driven attention. Although attentional shifting and attentional focussing are partially independent processes they are both mediated by the posterior attentional network

(Posner & Peterson, 1990). However, there was also evidence for differences in processing within the frontal attentional network, such that poor decoders did not show the same modulation of N2 amplitude by flanker compatibility.

Experiment 3 aimed to examine differences between good and poor phonological decoders in the allocation of attention to global and local levels of hierarchical stimuli. Poor phonological decoders were slower than good phonological decoders when attention was directed to both the global and local levels of hierarchical stimuli. This was accompanied by a lack of task-related modulation of the posterior N1 component and the N2 component at temporal sites. These findings indicate differences in the allocation of spatial attention and bottom-up perceptual filtering respectively. It is possible that poor decoders made greater use of the later perceptual filtering stage (N2) due to the inefficient allocation of spatial attention as evidenced by N1 amplitude.

Experiment 4 investigated covert orienting among good and poor phonological decoders. Poor phonological decoders showed fewer RT benefits for valid spatial cues relative to good decoders, particularly for LVF trials. This effect was greatest for male phonological decoders, who also showed a lack of N1 modulation in the RH for LVF trials and an overall lack of attentional modulation of N1 latency. Good decoders also showed a RVF RT advantage accompanied by greater involvement of the LH relative to poor decoders, particularly female good decoders. In contrast, female poor decoders showed greater involvement of the RH which may reflect compensatory processing. Although further research is required to assess the reliability of the sex differences observed in this study, preliminary evidence suggests that spatial attention deficits among poor readers are greater for males in comparison to females.

Experiment 5 aimed to investigate covert orienting during orthographic and phonological decision tasks. Consistent with early selection models of word recognition, good decoders showed consistent behavioural effects of spatial attention on word recognition. However, poor phonological decoders show less benefit of spatial attention particularly for

words presented in the LVF when processing was biased towards phonological processing and therefore required the left to right serial allocation of attention to individual letters within a word. These behavioural differences were accompanied by an absence of the attentional modulation of both P1 and N1 latency for poor decoders and the lack of N1 latency modulation was greatest for the phonological task. There was an overall reversal of the lateralisation of the N1 component. For words presented in the contralateral visual field, good decoders showed greater N1 amplitude at RH parietal sites and poor decoders showed greater N1 amplitude at LH parietal sites and a similar hemispheric reversal was found for N1 latency. Poor decoders showed a reduction in N1 amplitude at RH occipital sites for LVF valid trials and a lack of attentional modulation of P1 amplitude at RH parietal sites. Together these findings were generally consistent with a RH posterior parietal impairment accompanied by reliance on and/or less inhibition of the LH in poor phonological decoders.

Phonological Decoding, Spatial attention, and Dyslexia

Previous research findings suggest a link between nonword reading, spatial attention and developmental dyslexia (Facoetti et al., 2006; Kinsey et al., 2004), and according to familiarity sensitive models of spatial attention in reading (LaBerge & Brown, 1989), the involvement of spatial attention is at its greatest when the letters within words must be sequentially analysed as is the case during the phonological decoding of unfamiliar or nonwords. The present findings of spatial attention deficits among poor phonological decoders who were not necessarily diagnosed as dyslexic, strongly suggest a link between spatial attention and phonological decoding. Depending on the typology employed, the poor phonological decoders in the present research were likely to be most similar to those diagnosed with developmental phonological dyslexia (Coltheart et al., 1993) or dysphonesia (Boder, 1971), and depending on impairment on other reading measures (including orthographic tasks), may also be similar to mixed dyslexics or dysphoneidetics respectively. Furthermore, although many participants in the present study had

not received an official diagnosis of dyslexia, they typically differed from good decoders on several other reading measures including word identification, reading accuracy, reading rate, comprehension and vocabulary, suggesting considerable reading difficulty. This is consistent with the proposal that poorly formed phonological mechanisms are associated with a variety of the reading problems experienced in dyslexia (see Bradley & Bryant, 1983; Rack et al., 1992; Stanovich, 1988; Stanovich & Siegel, 1994; Wagner & Torgesen, 1987). However, it should be noted that poor decoders also differed from good decoders in terms of orthographic skill as assessed by an irregular word reading test, suggesting some similarities with dysideisia, or developmental surface dyslexia. However, the findings of Experiment 5 indicated that differences between good and poor decoders in terms of directing attention to the LVF were greatest for the phonological relative to the orthographic decision task. These findings provided clear support for the importance of spatial attention during phonological processing and were accompanied by a lack of attention-related modulation of N1 latency which was also greatest for the phonological decision task.

Evidence for Atypical Cerebral Lateralisation?

In the present series of experiments there was evidence for processing differences in both the RH and LH. Although these experiments aimed to assess mechanisms of spatial attention which is predominantly mediated by the RH, the letter and word stimuli used in these paradigms also required some language processing and therefore processing within the language dominant LH. Further, although the LH is also involved spatial attention it has been argued that the RH contributes to or mediates the control of spatial attention in the LH (Hillis et al., 2005; Kinsbourne, 1970).

The findings of several of the electrophysiological experiments in the present study were consistent with processing differences in the RH and therefore the RH deficit hypothesis of dyslexia (Facoetti et al., 2003a; Facoetti et al., 2001; Hari & Renvall, 2001; Stein et al., 1989;

Stein & Walsh, 1997). The greater interference effect for incompatible flankers observed for poor decoders in Experiment 2 was associated with less modulation of the N1 component at RH posterior sites. Similarly in Experiment 5, poor decoders showed a reduction in N1 amplitude at RH parietal sites, and at RH occipital sites for LVF valid trials and a lack of attentional modulation of P1 amplitude at RH parietal sites. In both Experiments 4 and 5 behavioural differences were greatest for LVF trials, which is also consistent with differences in RH processing. In Experiment 4, these behavioural differences were greater for male poor decoders who also showed a lack of N1 modulation in the RH for LVF trials.

Consistent with the RH hypothesis of dyslexia, the findings of other electrophysiological studies have indicated processing differences between dyslexics and controls at RH sites. For example, controls but not dyslexics showed greater P2 amplitude over the RH occipital cortex in response to stimuli presented at a range of contrasts and spatial frequencies (Schulte-Körne et al., 1999). Further, dyslexics showed a reduction in N100 amplitude in response to nonwords but not words at RH central sites (Wimmer et al., 2002). Wijers et al. (2005) found that controls but not dyslexics showed greater P350 in the RH (~350ms) during the cue-to-target interval when attention was sustained to the LVF.

Previous electrophysiological research has also indicated a reduction in ERP amplitudes in the LH among dyslexics. For example, dyslexics show a reduction N1 amplitude in the LH in response to contrast reversal stimuli (Hennighausen et al., 1994), during passive viewing of words and light flashes (Conners, 1970; Preston et al., 1974; Symann-Louett et al., 1977). A similar left lateralised reduction in amplitude has been observed for the P100 component (Brandeis et al., 1994) as well as later endogenous components (Harter et al., 1988b; Miles & Stelmack, 1994; Preston et al., 1977). In the present research, there was limited evidence for a LH processing deficit. However, in Experiment 4, good decoders also showed a RVF RT advantage accompanied by greater involvement of the LH relative to poor decoders. Further, at temporal and parietal sites good decoders showed greater N1 amplitude in the ipsilateral LH for

stimuli presented in the LVF in comparison to poor decoders. In Experiment 2 there was also some evidence for differences in attentional processing within the LH such that good decoders showed bi-hemispheric modulation of N1 amplitude by cue size and this modulation was only present in the RH for poor decoders.

However, in Experiments 2 and 5 the reduction in N1 amplitude at RH parietal sites for poor decoders was accompanied by greater activity in the LH and in Experiment 5 a similar reversal was found for N1 latency. This reversal in hemispheric asymmetries among poor phonological decoders could be due to compensatory activity. However, it is also possible that these differences occurred due to a lack of attentional control over the LH due to a RH deficit. RH areas of the PPC are proposed to contain bilateral receptive fields that represent both visual hemi-fields, whereas the LH has contralateral receptive fields that represent only the RVF (see Hillis et al., 2005). It has been suggested that RH temporo-parietal lesions cause inactivation of the right dorsal network, and according to hemispheric models of visual orienting (Kinsbourne, 1970) this inactivation may also result in relative hyper-activation of the LH dorsal network. Hyper-activation in the LH is also consistent with previous findings of over-distractibility in the RVF in dyslexia (Facoetti & Turatto, 2000; Hari et al., 2001). The reduction in P1 latency observed in the LH for poor decoders when words were presented in the RVF (Experiment 5) also supports this theory.

Some other researchers have found a reversal in hemispheric asymmetries of some ERP components for dyslexics relative to controls. In contrast to the present findings, this research has indicated a reduction in LH activity accompanied by greater involvement in the RH among dyslexics (Barnea et al., 1994; Jones & Michie, 1986; Lovrich et al., 2003). However, these researchers have typically investigated later endogenous components of the ERP waveform and have not typically employed tasks which tap RH mechanisms of spatial attention.

Some research findings indicate that hemispheric differences in electrophysiology are specific to particular subtypes of dyslexia. For example, in an EEG study it was found that

dyseidetics and dysphonetics show an over-reliance on the LH and RH respectively which is consistent with a compensation-from-strength model rather than the direction of difference proposed by Boder (Flynn et al., 1992). In contrast, McPherson et al. (1996) found that phonetics and dysphonetics showed greater N1 amplitude at RH and LH sites respectively. This was accompanied by few behavioural differences suggesting that these differences may have occurred due to compensatory strategies. These latter findings are consistent with the greater N1 amplitude observed in the LH for poor phonological decoders in the present study.

Whereas some of the differences reviewed so far between the present research and the dyslexia literature may be due to factors such as task demands (spatial vs. linguistic), compensatory processing including the impact of age differences, and the ERP components studied (exogenous vs. endogenous), it is also likely that sex differences play an important part in the differences observed. For example, findings in support of the RH deficit hypothesis were more common among males in this series of experiments, and females showed the most evidence for a LH linguistic deficit. This preliminary evidence for sex differences in hemispheric lateralisation and their relationship to the findings of the present thesis are discussed in further detail below.

Sex Differences in Hemispheric Lateralisation

A common finding in the neuropsychological literature is that males perform better than females on spatial tasks, whereas females perform better than males on verbal tasks (see Gur et al., 2000; Shaywitz et al., 1995). Considering that the left and right hemispheres are implicated in verbal and spatial tasks respectively these sex differences are thought to reflect differences in hemispheric organisation (Frith & Vargha-Khadem, 2001; Hiscock, Israelian, Inch, Jacek, & Hiscock-Kalil, 1995; Levy & Heller, 1992; Pugh et al., 1997; Rilea, Roskos-Ewoldsen, & Boles, 2004; Rossell, Bullmore, Williams, & David, 2002; Shaywitz et al., 1995). For example, during phonological processing, females have been shown to out perform males and show bilateral

activation in the inferior frontal gyrus (Shaywitz et al., 1995). In contrast, males showed lateralised activation in this area. Similarly, unilateral brain damage to the left hemisphere has been shown to result in reading and spelling impairment for males but not females (Frith & Vargha-Khadem, 2001), supporting the hypothesis that males show greater left hemisphere lateralisation for language in comparison to females. In contrast, for spatial tasks, males generally show better performance (e.g., Rilea et al., 2004) and show greater right hemisphere activation in regions associated with spatial processing in comparison to females (Gur et al., 2000). There is also some evidence that males show a similar “bilateral advantage” for spatial tasks, characterised by greater left hemisphere activation relative to females (Gur et al., 2000). Consistent with this theory, males also show greater L>R asymmetry and greater volume of the LH PPC in comparison to females (Frederikse, Lu, Aylward, PBarta, & Pearlson, 1999).

Together these findings are consistent with the suggestion that males show greater language specific hemispheric specialisation in comparison to females, which may result in a greater risk of experiencing dyslexia due to reduction in overall verbal ability or a reduction in their ability to compensate for damage in LH areas or unfavourable hemispheric asymmetries (Hier, 1979). In contrast, women may be better equipped to shift language functions to the RH due to greater bi-hemispheric language processing (Hier, 1979). Considering that males also show greater activation of the RH during spatial tasks, as well as greater LH involvement relative to females, damage to parietal areas of the LH or RH could affect spatial attention in males. It is also possible that males rely more on spatial attention during reading. However, this hypothesis requires empirical investigation.

Male vulnerability to reading disability is a widely reported but controversial finding. For example, the higher prevalence of males among dyslexic populations could be due to ascertainment bias produced by subjective diagnosis methods. However, the findings of a recent meta-analysis suggest that when ascertainment biases are minimised, there is still a greater proportion of dyslexic males than females, with an estimated gender ratio of 1.74-2.00

(Liederman et al., 2005). Leiderman et al. (2005) concluded that gender differences should be considered as another variable that defines reading disability.

Despite well documented sex differences, the majority of studies investigating lateralisation or hemispheric organisation in dyslexia have failed to make a distinction between males and females, and many studies have been restricted to male populations only. While the present research was not designed to investigate sex differences, and thus did not aim to recruit equal numbers of males and females, sex was included as a factor in some analyses and was reported where appropriate. However, a greater number of females were recruited relative to males and findings in relation to males should be interpreted with caution. Further, although the prevalence of dyslexia is reported to be higher among males relative to females, the prevalence of females is reported to be higher among compensated dyslexics (Lefly & Pennington, 1991).

In addition to overall differences in the lateralisation of ERP components in the present study, such that RH differences were greater for males and LH differences were greater for females, opposite reversals in hemispheric asymmetries were observed for female and male poor decoders, particularly at temporal sites. In Experiment 2 there were overall differences in hemispheric lateralisation at temporal sites, such that female poor decoders showed a reduction in N1 amplitude in the LH relative to female good decoders, male poor decoders showed a reduction in the RH relative to male poor decoders, and female poor decoders showed compensatory processing in the RH.

In Experiment 3, female poor decoders and male good decoders showed greater N1 amplitude at RH relative to LH temporal sites and relative to female good decoders and male poor decoders. Similarly, female good decoders and male poor decoders showed greater N2 amplitude at LH relative to RH temporal sites and relative to female poor decoders and male good decoders. For males there were also trends for lateralisation effects on N1 latency at ipsilateral occipital sites, such that male poor decoders tended to show shorter latency in the LH (LVF) and male good decoders tended to show shorter latency in the RH (RVF).

As mentioned in the previous section, in Experiment 4, it was male poor decoders who showed a lack of N1 modulation in the RH for LVF trials and an overall lack of attentional modulation of N1 latency. In contrast, female good decoders showed a reduction in LH activity and greater involvement of the RH which may reflect compensatory processing. Although there were not enough male subjects to include sex as a factor in Experiment 5, it is also possible that sex differences contributed to the hemispheric differences observed in this experiment.

Although further research is required to assess the reliability of the sex differences observed in the present experiments, preliminary evidence suggests that spatial attention deficits among poor readers are greater for males in comparison to females and whereas males show greater processing differences in the RH, the opposite is true for females. Further, female and male poor decoders may also show compensatory processing in the RH and LH respectively.

Compensatory processing

The poor phonological decoders recruited in the present series of experiments were functioning within a university population and were likely to have developed strategies to compensate for poor phonological decoding skills (Bruck, 1990; Nation & Snowling, 1998; Snowling et al., 2000; Torgesen et al., 2001). Previous neuroimaging research has shown that some dyslexics develop compensatory strategies that involve the RH and frontal areas such as the anterior frontal gyrus (Brunswick et al., 1999; Pugh et al., 2000; Richards et al., 1999; Salmelin et al., 1996; Shaywitz et al., 1998; Simos et al., 2000). In the present research evidence for compensatory processing among the poor phonological decoders was greater for females relative to males and poor decoders also showed greater activity in the LH relative to RH under some circumstances. This discrepancy may be due the fact the tasks used in the present study emphasised spatial attention mechanisms that are mediated by RH parietal areas.

There was also evidence that poor phonological decoders compensate for early attentional processing difficulties through differences in later processing stages. For example, in

Experiment 3 poor decoders did not show the same reduction in N1 and N2 amplitude for the global task that was observed among good decoders, but they tended to show greater N2 amplitude in comparison to good decoders overall. The posterior N2 is thought to reflect a non-spatial filtering process involved in isolating one local item from another (Han & He, 2003). Thus the greater N2 amplitude observed for poor decoders may reflect compensatory processing at a later stage due to a deficiency in early spatial selection.

Evidence for a magnocellular deficit?

The present research did not specifically aim to examine the functioning of the magnocellular system. However, spatial attention deficits among developmental dyslexic populations have often been interpreted as representing a deficit in the PPC which receives predominantly magnocellular input. Further, the findings of Experiment 1 indicate that the performance decrement observed for poor decoders during visual search were greatest for the form/motion task in which the involvement of the M and P pathways were maximised and reduced respectively. The identification of flanked letter is also thought to involve the magnocellular system (Omtzigt & Hendriks, 2004; Omtzigt et al., 2002) and it is therefore possible that differences in magnocellular processing contributed to the differences observed between good and poor decoders in Experiment 2. In Experiment 3 poor decoders tended to be less accurate for the global task. This data does provide some support for the hypothesis that an M impairment would selectively impair global processing in poor readers (Keen & Lovegrove, 2000), but this finding should be interpreted with caution as the mean accuracy of both groups was over 95%. Future research employing spatial attention manipulations within tasks that preferentially stimulate the M and P pathways is required to fully investigate M pathway involvement in spatial attention deficits among poor decoders and developmental dyslexics.

Summary and Conclusions

The present series of experiments has shown that adults with poor phonological coding skills differ from good phonological decoders on tasks that require control over spatial attention mechanisms subserved by the posterior attentional system. Poor phonological decoders were slower to search for feature conjunctions in visual search tasks, were less able to filter distracting flanker stimuli, and to sustain attention to either the local or global levels of hierarchical stimuli. Further, poor phonological decoders showed fewer benefits of valid spatial cues during covert orienting tasks, and a disruption of spatial cueing in the LVF for both traditional covert orienting paradigms and particularly when making phonological rather than orthographic decisions during cued lexical tasks. These findings are broadly consistent with previous research reporting spatial attention deficits among dyslexic populations and suggest a deficit in processing within RH parietal areas. Few previous studies have linked spatial attention deficits in dyslexia to the modulation of attention related components of the ERP waveform. Experiments 2-5 of the present thesis showed that the behavioural deficits observed among poor phonological decoders were consistent with differences observed in the modulation of the P1 and N1 components of the ERP waveform, particularly in the RH. There was some preliminary evidence for sex differences in that overall reversals in hemispheric asymmetries among poor decoders were observed in the LH among females and the RH among males. However, further research is required to clarify the relationship between phonological decoding, sex differences, and hemispheric lateralisation. The findings of the present research indicate the utility of this methodology for future investigations of spatial attention deficits among dyslexic populations in order to link behavioural findings with underlying electrophysiology.

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APPENDIX A

Irregular word reading test

Me	Circuit	Schism
Is	Tongue	Beret
No	Tow	Indict
The	Chorus	Regime
Was	Lose	Quay
One	Cough	Benign
Good	Sword	Ninth
Give	Ton	Bough
Come	Routine	Righteous
Eye	Yacht	Heirloom
Wolf	Choir	Caste
Work	Champagne	Epoch
Head	Drought	Khaki
Friend	Brooch	Valet
Pretty	Tomb	Gross
Shoe	Nought	Stead
Break	Foreign	Schedule
Bowl	Distraught	Covet
Sugar	Plover	Stoic
Touch	Bouquet	Chorale
Answer	Sovereign	Deity
Soul	Trough	Niche
Island	Depot	Clerk
Blood	Colonel	Fjord
Iron	Scythe	Wrath
Sure	Gauge	Mauve
Busy	Debris	Plaid
Stomach	Meringue	Bureau
Ceiling	Pint	Encore

APPENDIX B

Orthographic and phonological decision task stimuli

Orthographic Decision Task		Phonological Decision Task	
Real Words	Pseudohomophones	Pseudohomophones	Nonwords
PRUNE	PREWN	SPIL	FLIL
SKATE	SKAIT	FLURT	THURT
GROAN	GRONE	CRUCH	CRONT
FROWN	FROUN	BATE	BOAP
YOLK	YOAK	THIE	BLIE
FOAL	FOLE	COAV	COAF
GERM	JERM	FRITE	FRYLE
BRINK	BRYNK	KIRL	KINE
SCARE	SCAIR	KLOAK	GLOAK
CLOWN	CLOUN	FLOTE	FLOAP
CHALK	CHORK	BROOZ	BROOL
CHEAT	CHEET	RUMB	TUMB
CLOAK	KLOAK	WHERM	GLERM
RHYME	WRIME	MOAL	MOIN
GRATE	GRAIT	HAWL	VAWL
SPICE	SPISE	KAUD	KEAM
SHINE	SHYNE	GHOAT	GHACK
GROOM	GRUME	COFF	POFF
GOAT	GOTE	PLANC	PIME
CORD	KORD	GLAIR	SLAIR
COMB	KOME	PYPE	PYME
STIR	STUR	SPEER	SPEAP
SWORD	SORDE	RECK	YECK
BEAST	BEEST	HEIL	HOIL
PLANK	PLANC	LEACE	BEASE
GLUE	GLEW	MEAK	VEEK
THIEF	THEAF	BIGHT	VIGHT
GLUE	GLOO	FAUGE	RAUGE
STEER	STEAR	TOOM	TOMP
HASTE	HAIST	CRAUL	CRAIL
RAID	RADE	LEEF	LEET
GRIEF	GREEF	KAKE	DAKE
STOLE	STOAL	KOAN	KEET
ZINC	ZINK	STEAP	STOOT
PORK	PAUK	SHIE	GRIE
TEAR	TAIR	HEEP	ZEEP
PLEA	PLEE	CLOO	SLOO
JUICE	JEUCE	ROAP	GOAP
ZONE	ZOAN	THRED	THRON
GHOST	GOAST	KNUT	GRUT
NERVE	NURVE	SOOP	DOOP
BLOOM	BLUME	SKECH	BRECH
BAKE	BAIK	FLUD	FLIM

ROAR	RORE	LEEN	HEEN
SHOE	SHUE	CREEM	DWEEM
KNOW	KNOE	BEEM	VEEM
PURSE	PURCE	SKRUE	SKRAT
SWORE	SWOAR	JOAK	JOPE
LAWN	LORN	KARD	CARN
TROOP	TRUPE	SHURT	SHART
SEAL	SEEL	PRYZE	PROOZ
FLEET	FLEAT	CLOWD	CLOME
DOME	DOAM	SERCH	DERCH
FAME	FAIM	LARF	LERF
DRAIN	DRANE	TITE	PITE
POLE	POAL	DORN	NORN
SAUCE	SORSE	FENSE	FELCE
SKIRT	SKERT	FAIT	NAIT
SCREW	SCRUE	BOAN	PONE
URGE	ERGE	SKEAM	CLEAM
SOAP	SOPE	FLYE	SMYE
SHAME	SHAIM	WROLE	CHOLE
BOWL	BOAL	CARM	RARM
PILE	PYLE	LAICE	SAICE
SHIRT	SHERT	KASH	MISH
GRAIN	GRANE	CHOES	CHISK
TRAIL	TRALE	TEECH	NEACH
BONE	BOAN	THRET	THOOF
CORN	KAWN	PAICE	WAICE
ROLL	ROAL	DELT	DERR
MYTH	MITH	BRANE	SMANE
WEAR	WAIR	THRU	THREP
HURT	HERT	SUTE	SULE
FOAM	FOME	SLITE	SKITE
CORE	KOAR	WRISC	WRALT
SMOKE	SMOAK	SEET	SEAF
COAT	COTE	GHESS	GHOLT
DIRT	DURT	BAIR	JAIR
COAT	KOAT	SAIF	SAIP
CURVE	KERVE	LIGH	LIRP
THREW	THROO	GROE	GRAT
MILE	MYLE	TRANE	TRAIF
COUNT	KOUNT	SPRED	SPREM
LAKE	LAIK	SHAIP	SHATE
WOOD	WOUD	WATE	NATE
WHEEL	WHEAL	SYNE	SYTH
HOLE	HOAL	CAUT	JAUT
SNOW	SNOE	FITE	FIPE
SCALE	SKAIL	WAWK	WAFF
GREW	GROO	BILT	VILT
SLEEP	SLEAP	SPEEK	SPOWL
SCORE	SKORE	DOWT	DOAN

BIRTH	BURTH	FEAD	FEEM
CHAIR	CHARE	GAIM	GOME
RAIN	RANE	PHEER	PLEAR
GAIN	GANE	RONG	DONG
STORE	STOAR	FREND	FRORE
TOKE	TOAN	TRIGH	BRIGH
WATCH	WHOCH	FLOAR	PLOOR
DRINK	DRINC	WAWL	DAWL
YOUTH	YOOTh	KAUL	KAIL
BANK	BANC	CAIVE	HAIVE
WAIT	WATE	THURD	THORD
CLAIM	KLAME	LEEVE	MEAVE
DATE	DAIT	KLASS	CLISS
DEEP	DEAP	RAIT	RABE
FIRM	FERM	GHERL	GHORN
STAFF	STARF	VOISE	VORNE
GREEN	GREAN	KEAP	TEEP
HORSE	HAUCE	TIRN	TURT
CHIEF	CHEEF	NAIM	NADE
WRONG	RHONG	CRIGH	GLIGH
BLUE	BLOO	KIGHT	KIDGE
FALL	FAUL	PRAIR	PRALP
TALK	TORK	FRATE	FRAKE
FINE	FIGN	STANE	GLANE
PEACE	PEESE	THAUN	PLAUN
FEEL	FEAL	GHATE	FLATE
KEEP	KEAP	NALE	CALE
WORD	WURD	CARF	NARF